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ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 56 SEPTEMBER, 1947 No. 3

Vertigo

SYMPOSIUM

BEFORE THE AMERICAN OTOLOGICAL SOCIETY

ST. LOUIS, MO., APRIL 22, 1947

The Physiology of the Vestibular Mechanism in Relation to Vertigo. William J. McNally, M.D., Montreal, Canada.

The Symptomatology of Vertigo. Alfred Lewy, M.D., Chicago, Ill.

Pathology of Vertigo of Peripheral Vestibular Origin. John R. Lindsay, M.D., Chicago, Ill.

The Incidence of Vertigo in Neurological Diseases. Irwin Levy, M.D., and James L. O'Leary, M.D., St. Louis, Mo.

Summation. Albert C. Furstenberg, M.D., Ann Arbor, Mich.

XLVIII

THE PHYSIOLOGY OF THE VESTIBULAR MECHANISM IN RELATION TO VERTIGO

W. J. McNally M.D.

Montreal, Canada

The words vertigo and dizziness are usually used interchangeably by clinicians. Occasionally an attempt is made to distinguish between the two. They derive from different languages but they both mean a turning sensation. For present purposes we shall accept them as synonymous. Vertigo may be defined as a consciousness of discord in the postural mechanism. Maintenance of posture depends upon information received from the labyrinths, from the eyes and from muscles and joints, the so-called kinesthetic sensations. This information is correlated in the consciousness, and conflicting information from any source or failure of the central nervous system to coordinate this information leads to confusion in the mind of the individual; this is what we call vertigo or dizziness. A patient frequently has great difficulty in describing his sensations. He may use the word dizziness to describe a variety of feelings. McNally and Stuart[45] reported the description by patients of their sensations following caloric labyrinthine stimulation. In many there was no sensation of turning. Faintness, unsteadiness, rocking, staggering, swimming, weakness, backward swaying, waviness were mentioned by different patients. It will be seen by this that vertigo includes a wide variety of sensations in addition to a turning sensation. Whereas maintenance of equilibrium is an incidental function of the ocular mechanism and the kinesthetic mechanism, it is the main function of the vestibule or the labyrinth. It is my task to outline briefly the significant facts in the physiology of the labyrinth which enable us to understand how its disturbance causes vertigo.

While reviewing the literature in preparation for this symposium, I was much impressed by a remark made by Fischer:[22] "Das wir es mit recht komplizierten Dingen zu tun haben." I agree that this is a very complex thing with which we are dealing and Lewes[35] says, "Whenever a complex question is answered by a neat formula pretending to embrace all the phenomena and to clear up all obscurities, it is a wise caution which suspects there is something

wrong." If you agree with Lewes, you will suspect nothing wrong with what I have to say here because I present no neat formula for the solution of this complex fascinating subject, the physiology of the labyrinth.

It is interesting to note that most of the information which we possess about the function of the labyrinth has been accumulated during the last one hundred years. Purkinje, in 1825, noted that eye reactions and dizziness may occur during and after rotation. About the same time Flourens operated on a pigeon's semicircular canals and noted that nystagmoid movements of the head and eyes occur in the plane of the injured canal.

In 1861 Ménière reasoned that dizziness is so often associated with aural disease—hearing loss and tinnitus—it is probable that the dizziness arises from disease or irritation of the semicircular canals.

Goltz[24] was the first to reason that since interference with the labyrinth causes dizziness, the normal function of the labyrinth is to control equilibrium.

Mach,[42] Breuer[7] and Crum Brown[12] quite independently of one another in 1875 stated that it is the flow of endolymph which is the adequate stimulus of a semicircular canal and ampulla. It was Breuer who first connected nystagmus during and after turning in both animals and man with stimulation of the semicircular canals. Breuer stated that whereas the canals are stimulated by rotatory movements, the otoliths are stimulated by movements of the head out of the horizontal plane and by linear or progressive movement.

Ewald[21] carried out an ingenious experiment upon the semicircular canals which is a classic. By sealing off a semicircular canal of a pigeon and inserting a pneumatic hammer between the blocked area and the ampulla, he was able to show that endolymphatic flow in one direction through the ampulla is a greater stimulus than in the other direction. He showed that whereas flow towards the ampulla in a horizontal canal is the maximal stimulus, flow away from the ampulla is the maximal stimulus in the vertical canals.

These findings of Ewald have been generally accepted to explain the mechanism of canal stimulation in the human labyrinth and his findings have had the force of laws. During recent years some experiments have been reported which suggest that these laws require some modification when applied to the mammalian labyrinth.

Lee,[34] Laudenbach,[33] Kubo,[32] Maxwell,[44] McNally and Tait,[46] Steinhausen,[63] Ross[56] and others have found that in the lower animals Ewald's law must be modified because a semicircular canal in the fish and the frog responds to stimulation in only one direction and that is in the direction of the maximum stimulus pointed out by Ewald. There is no reaction in the direction of the so-called minimal stimulus.

Bárány[1] gave the credit for developing the rotation or turning test to Kreidl, James and Wanner. This was the chief clinical test of labyrinthine function until Bárány described the caloric test. The advantage of the caloric test over the rotation test is that with it one can study each labyrinth separately.

Magnus and deKleyn[43] studied labyrinthine function and placed the labyrinth in its true relationship with the central nervous system by analysing and clarifying labyrinthine reactions and by describing related postural and righting reflexes of nonlabyrinthine origin.

In 1922 Kobrak modified Bárány's caloric test and showed that a minimum caloric stimulation elicits more information about the state of the labyrinth than does a maximum stimulation.

Maxwell[44] showed a close relationship between the semicircular canals and otolithic function in the dogfish.

Tait and McNally[46] carried out uncomplicated ampullary nerve section in the frog and showed that a semicircular canal elicits reactions of the head and body musculature necessary to compensate for any head movement in the plane of that canal. They[67] also reported a close relationship between the vertical semicircular canals and the utricles.

Steinhausen[63] succeeded in visualizing the cupular movements in the semicircular canal ampulla in the live pike and thus was able to prove experimentally Mach's, Breuer's and Crum Brown's theory of semicircular canal stimulation. He was able to show that rotatory and post-rotatory nystagmus result from deflections of the cupula in the ampulla.

Ross[56] recorded action currents from the individual nerves of the semicircular canals in the frog and confirmed the theory that the semicircular canals are stimulated by angular acceleration. He confirmed Steinhausen's experiments that it is the deflections of the cupula that are signalled to the brain and which are responsible for

the nystagmus resulting from the stimulation of the semicircular canals.

The weight of experimental evidence demonstrates that the saccule is not concerned with equilibrium but that it may be affected by vibration stimuli.

Kreidl,[31] Maxwell[44] and Ulrich[70] all reported experiments in which an attempt was made to stimulate the utricle directly. The animals used were live fish. Ulrich's experiments were probably the best controlled. Movements of the otolith of the utricle elicited compensatory movements of the eyes but there was no nystagmus following any of these stimulations. The experiments of Maxwell,[44] Magnus and deKleyn (See Magnus[43]), Versteegh,[71] Tait and McNally[67] and Ross[56] all indicated that the utricles are not stimulated by rotation about a vertical axis. The experiments of McNally and Tait showed, however, the utricles in the frog are indirectly concerned during the reaction to rotation about a vertical axis because in the absence of the utricles the head is no longer held level and there may then result some stimulation of the vertical canals. Slow tilting about a horizontal axis stimulates the utricles as shown by the experiments of Maxwell, Tait and McNally, Versteegh, and Ross. Tait and McNally reported that the frog's utricle can be stimulated by a quick tilt (angular acceleration) and that the reaction is anticompensatory. They reasoned that this reaction counteracts those of the semicircular canals and tends to hold them in check. Ross who recorded the action currents from the utricle in the frog was not able to detect any indication of this anticompensatory reaction from the utricle in response to a quick tilt.

Magnus and deKleyn centrifuged away the otolithic membranes of the guinea pig by the method of Wittmaack and they concluded that the utricles controlled the attitudinal reflexes, the symmetric labyrinthine righting reflexes and possibly the rotatory compensatory positions of the eyes.

Versteegh[71] was able to sever the nerve to the utricle on one side in the rabbit and he found that all the positional reflexes in response to rotatory movements to the operated side disappeared (including those previously allocated to the saccule).

Tait and McNally[67] were able to carry out bilateral utricular nerve sections in the frog. They showed that after severing the nerves to both utricles the frog no longer responded to centrifugal

force or to linear acceleration; whereas a bisoliutricular frog (nerves to all other labyrinthine end organs severed except to the utricles) showed normal responses to both. The utricle is the most important gravity organ in the labyrinth. It is stimulated by slow tilting about a horizontal axis, by centrifugal force and by linear acceleration. It may also under certain conditions be stimulated by angular acceleration. It elicits all the positional labyrinthine reflexes of the body and the eyes.

Nystagmus. Borries[6] reported that Darwin studied and wrote about rotatory and post-rotatory nystagmus in 1800. As stated before Breuer[7] was the first to associate rotatory nystagmus with labyrinthine stimulation. Ewald[21] and Steinhausen[63] supplied the experimental evidence that the semicircular canals possess a mechanism for the production of nystagmus. Spiegel's[60] experiments showed that both the slow and quick phases of nystagmus arise most probably within the vestibular nuclei. It has been repeatedly shown, especially by Magnus and deKleyn, that the only part of the central nervous system essential for nystagmus is that part of the brain stem from below the vestibular nuclei to just above the oculomotor and trochlear nuclei. The forebrain has an influence on nystagmus but is not essential to it. Lorente de Nó and deKleyn both demonstrated that intimate relations exist between the central nervous system, the labyrinths and the individual groups of eye muscles involved in nystagmus. Magnus and deKleyn, Versteegh,[71] Hasegawa,[28] and Ulrich[70] have concluded from their experiments that stimulation or injury of the utricle does not elicit nystagmus.

Clinically it has been found that nystagmus may arise spontaneously and that the nystagmus may be caused by disease or irritation in the ocular mechanism, in the central nervous system, or in the labyrinth. This spontaneous nystagmus in some cases may only occur when the head is in a certain position. This last nystagmus is referred to as positional nystagmus. It was described by Bárány in 1921. Nylén[53] has classified positional nystagmus into two types and these are confirmed by Lindsay. In Type 1 the direction of the nystagmus changes with a change of the position of the head. It occurs predominantly in central nervous system lesions. The case may be one of cerebellar tumor or of multiple sclerosis. Lindsay found Type 1 nystagmus in 29 of a series of 30 cases of pseudo-Ménière's syndrome (in which auditory disturbance and central neurological signs were absent). In Type 2, the direction of the nystagmus does not change with changes in position of the head, but the nystagmus may appear only in certain positions or it may be in-

fluenced in intensity by the position of the head. This type of nystagmus may be found either in peripheral lesions or in central lesions. It may be found in a case of cerebellopontine angle tumor, in a case of concussion involving the central mechanism, or in true Ménière's syndrome.

A special examination must be made in order to uncover positional nystagmus. It has not been definitely determined whether the cause of positional nystagmus is a lesion within the labyrinthine end organs or whether it is the result of some lesion within the central nervous system. deKleyn and Versteegh[16] have suggested that in some cases it is the result of the position of the head affecting the circulation to the brain. It may be a matter of neck reflexes resulting from the peculiar position of the head in relation to the trunk which is affecting the eye movements. It has been suggested that the utricle is the most likely labyrinthine end organ responsible for positional nystagmus. This is concluded because experimentally the utricle is stimulated by tilting the head out of its normal, level horizontal position. As stated before there is no experimental proof that utricular stimulation causes nystagmus. The positions best suited to elicit positional nystagmus suggested by Nylén and Lindsay are head shaking, stooping and rising up, sitting up quickly, and after tests for excitability. In addition to these the patient is examined lying on the left side, lying prone, lying supine and lying with the head hanging back over the end of the table. Lindsay[36] stated that the detection of a positional nystagmus (Type 1) is of assistance in diagnosing pseudo-Ménière's syndrome and he believes that this positional nystagmus indicates a central origin for pseudo-Ménière's syndrome.

Some cases of atypical Ménière's syndrome are really only early cases of true Ménière's syndrome before the development of the classical symptom triad—dizziness, tinnitus and deafness. In these cases, of course, the disturbance is in the peripheral labyrinth. Lindsay's series contained cases of atypical Ménière's syndrome probably the result of cerebral vascular lesions and they would not subsequently become cases of true Ménière's syndrome.

The examination for positional nystagmus is particularly important when a patient complains that his dizziness is more marked in a certain position of the head as, for instance, when he puts his head back or when he lies down or when he turns towards one side while in bed. If a patient is examined in the position in which he complains of being uncomfortable, it may be found that he has a

positional nystagmus while in this position and this is objective evidence of his subjective disturbance.

Dizziness. Dizziness is the consciousness of disordered sensations and must have essential pathways through the hindbrain, midbrain and forebrain. Penfield and Gage[54] reported that during brain operations on some patients they were able to elicit vertigo from stimulation of the cortex of the first temporal convolution. This is close to the auditory center.

It has been well established that there is a very rich connection between the vestibular nuclei and other parts of the brain. Though much anatomical and histological investigation has been carried out, the exact pathways in many instances are not known. It is this lack of detailed knowledge which renders the information derived from the vestibular tests of questionable value in intracranial diagnosis.

Dizziness or vertigo may result from stimulation, irritation or disease of any of the structures concerned in the maintenance of balance. We, therefore, may have ocular vertigo, central vertigo or labyrinthine vertigo. Frequently, it is difficult to distinguish between the various causes. We must rely upon the history and the description of the vertigo and upon the associated signs and symptoms.

In the case of ocular vertigo there are usually other signs of ocular disease and as a rule the vestibular tests are normal.

In the case of central vertigo, distinction between this and labyrinthine vertigo is not easy. Frequently there are associated signs of central nervous system disease. In both central and ocular vertigo, if there is associated nystagmus, it is usually of long standing. If the disease is central and supratentorial, vestibular tests may reveal what deKleyn, Dusser de Barenne and Versteegh have called a nystagmusbereitschaft. This will be described in greater detail later. If the disease is subtentorial (located in the brain stem), the nystagmus resulting from labyrinthine stimulation may show perversion or dissociation. That is, the eyes do not move simultaneously and conjugately or the direction of the nystagmus or the plane of the nystagmus may change from what would normally be expected. There may be spontaneous vertical nystagmus. There may be positional nystagmus. McNally and Stuart[45] reported finding dizziness as a symptom in ten per cent of 600 consecutive cases admitted to the Montreal Neurological Institute over a six months' period.

If the cause of the dizziness or vertigo is within the labyrinth, there is frequently associated evidence of disease of the cochlear

mechanism as instanced by the presence of deafness and tinnitus. The vestibular tests may or may not be abnormal. There may be spontaneous or positional nystagmus.

Crowe[11] reported that the vestibular tests were normal in both ears in 35 per cent of 117 cases of Ménière's syndrome. DeKleyn and Versteegh[17] reported normal findings in 16 per cent and an unequal sensitivity in the two labyrinths in 73 per cent. Cawthorne and Hallpike[10] reported normal findings in 12 per cent of cases of Ménière's syndrome and they reported a canal paresis on the affected side in 49 per cent. Crowe reported subnormal vestibular reaction on the affected side in 19 per cent and no reaction on the affected side in 29 per cent. DeKleyn and Versteegh reported a nystagmusbereitschaft or directional preponderance in 11 per cent and Hallpike and Cawthorne reported a nystagmusbereitschaft in 21 per cent. The latter reported a mixed directional preponderance and canal paresis in an additional 19 per cent of their cases.

Directional Preponderance. Dusser de Barenne and deKleyn[20] found after removal of a cerebral hemisphere in the rabbit that any labyrinthine stimulation which caused a nystagmus towards the side of the cerebral lesion elicited a greater reaction than a stimulus which elicited a nystagmus in the opposite direction. They called this a nystagmusbereitschaft. Hallpike and Cawthorne have suggested the English name of directional preponderance. DeKleyn and Versteegh[71] found a directional preponderance in causes of brain tumor in man. Vogel[72] described it in cases of posterior fossa tumor, mutiple sclerosis and syringobulbar disease as well as in Ménière's syndrome of traumatic and nontraumatic origin. Fitzgerald and Hallpike[23] localized the brain lesion and reported that this directional preponderance occurred only in cases of temporal lobe lesions. Grahe[26] described directional preponderance to the side of the lesion in 11 per cent. Cawthorne and Hallpike[10] have reported a series of 100 cases of Ménière's syndrome and directional preponderance to the affected ear was present in 21 per cent of the cases.

Cawthorne, Fitzgerald and Hallpike[8] have attempted to explain the directional preponderance as being of vestibular origin. They have considered the whole problem of spontaneous nystagmus which follows destruction of one labyrinth.

They concluded that because the nystagmus lasts for some time after labyrinthectomy and since also the nystagmus is continuous at rest, the impulses concerned must be of the tonic class and must,

therefore, be chiefly of utricular origin. They give some evidence to show that the utricle is the labyrinthine end organ most concerned with labyrinthine tone. They argue that the compensatory or slow phase of nystagmus is tonic and therefore is utricular in origin so that postlabyrinthectomy spontaneous nystagmus is utricular in origin.

They then report a series of cases in which the labyrinth was surgically removed for treatment of Ménière's syndrome. In these cases they found that, when spontaneous nystagmus to the side of the intact labyrinth subsided, it was immediately followed by a directional preponderance to the same side. They concluded that because the directional preponderance follows the spontaneous nystagmus in such rapid succession and since it is directed to the same side it also must be a utricular reaction.

Utricle. Following a unilateral labyrinthectomy an animal's head and body are maintained in a special asymmetrical position. This position was well described by Ewald[21] in the case of the pigeon and the frog. In some animals this position is maintained for a much longer time than in others and the maintenance of the position is usually ascribed to the utricle. From this it has been argued that the utricular macula under these conditions is constantly stimulated and, therefore, is sending out a continuous flow of impulses to the central nervous system. Tait and McNally[67] pointed out that Beritoff[4] showed that in the case of the frog the asymmetrical position which follows unilateral labyrinthectomy could be undone by gently grasping the frog's head and slowly rotating it back into symmetry. If the frog's head is then gently released, the symmetrical position is maintained for some time. It is movement of the head which undoes the symmetry and a head movement stimulates the labyrinthine receptors. Tait and McNally pointed out in their experiments with frogs whose labyrinth had been completely removed that the muscle receptors themselves are adequate to hold any posture that has once been successfully imposed. So long as an animal's head remains motionless, there is no stimulation of the labyrinth, no labyrinthine excitation and no labyrinthine reflex. Tait and McNally concluded that the organs of the labyrinth concerned in reflex adaptation of bodily posture to the field of gravity are the utricular maculae. It is clear, nevertheless, that their function cannot be the steady holding of posture. Their part is presumably, on the occurrence of some form of otolithic displacement, promptly to break up an existing postural set and as promptly to impose one that is physiologically more suitable to the changed inclination of the substratum. If this conception of

utricular action is correct, the utricle is not necessarily the cause of positional nystagmus or postlabyrinthectomy nystagmus simply because the nystagmus occurs while the head is at rest. If the nystagmus occurs while the head is at rest, it is a reason why the nystagmus may not be utricular in origin.

As stated previously the experiments of Kubo, Hasegawa, Maxwell, Ulrich and Versteegh did not elicit any nystagmus from utricular stimulation even though there is a close association between the semicircular canals and the utricle.

The following three sets of experiments would seem to show a direct connection between nystagmus and utricular stimulation but all three of these need confirmation.

Borries[5] removed all three semicircular canals in the pigeon and he reported that he was still able to elicit normal postcaloric nystagmus by douching the external meatus with hot or cold water, and by changing the position of the head he could reverse the direction of the nystagmus. He concluded that in these cases the caloric reactions must arise from the maculae of the vestibule or from the macula lagenae. These experiments should be repeated before they are accepted because the great bulk of experimental evidence would indicate that the presence of the intact semicircular canals is essential to normal postcaloric nystagmus.

Thornval[69] plugged a semicircular canal in the pigeon after the method of Ewald but instead of making a second opening between the plug and the ampulla he made an opening between the plugged area and the non-ampullated end of the canal. When he exerted pressure on a canal fistula in this area, the nystagmus which he elicited was invariably in the plane of the anterior vertical canal on the same side irrespective of which canal he stimulated. Suction or aspiration on such a fistula had no effect and the nystagmus was not influenced by changing the position of the animal's head in space. If he removed the ampullae of the canals, the reaction disappeared in the majority of cases but in a few isolated cases there was a feeble but unmistakable reaction. In spite of this, however, he concluded that he was probably stimulating the utricle. In another series of pigeons, when he removed all three ampullae and semicircular canals, he could not elicit any nystagmus from cold water irrigation of the ear. He concluded that the caloric reaction in pigeons originates chiefly in the semicircular canals. He stated that the otolithic organs may be susceptible to caloric stimulation or may have an influence on

caloric nystagmus but in his opinion the caloric reaction in pigeons is chiefly brought about in the semicircular canals. His results are somewhat confirmatory of those of Borries.

Wittmaack[73] described an experiment in which he inserted a cannula through the oval window and when he exerted pressure through the cannula, he elicited a nystagmus in one direction. If he inserted the cannula further into the vestibule, the direction of the nystagmus was reversed and he concluded that this was the result of utricular stimulation. His experiments require confirmation.

The next two experiments are well controlled and would indicate that the utricle is not essential to nystagmus.

DeKleyn and Lund[15] reported a series of experiments in which they carried out simultaneous bilateral cold caloric stimulation of the ears in rabbits and during stimulation they examined the animals for compensatory reactions to change in position of the animal's head. These eye and limb reactions, the result of utricular stimulation, remained normal in spite of the simultaneous stimulation of the semicircular canals by the cold water. In another set of experiments they removed the otolithic membranes from both labyrinths in guinea pigs by the centrifuge method of Wittmaack. They subsequently found that the caloric nystagmus in both labyrinths remained normal and unchanged and that the caloric reactions could still be effected by change of the position of the animal's head in space as is the case in normal animals.

These two sets of experiments would indicate that on the one hand caloric stimulation does not interfere with the compensatory eye reactions which are known to follow utricular stimulation and, on the other hand, absence of the utricular otoliths does not in any way interfere with the normal postcaloric reaction from the semicircular canals. Such well controlled experiments as these do not indicate that nystagmus arises from utricular stimulation.

VESTIBULAR TESTS

Clinical tests for examination of utricular function have never received general clinical application.

The Slow Tilt Test. The idea of slowly tilting the patient out of the horizontal plane as a means of determining utricular function was first suggested as a clinical test by Jones and Fischer[29] and by Grahe.[25]

Grahe has described two tests for utricular function. One test consists of strapping the patient to a movable frame and tilting him from a position out of the vertical into the vertical with his eyes closed. The patient is asked to signify when he is in the vertical position. If he cannot detect the vertical position, it is concluded that the utricles are at fault. A second test consists of tilting a patient about a horizontal axis out of the vertical plane. The patient is strapped to a frame but his head is free to move and his eyes are closed. Normally the head should maintain the erect position. If the utricles are abnormal, the head moves with the body and is not maintained in the erect position. These tests have not received general acceptance.

The Rapid Tilt Test. The rapid tilt test was introduced into the laboratory by Ewald[21] and Thomas.[68] It was first suggested as a clinical test by Tait and McNally (see Tait[66]). The quick test is primarily a test of vertical semicircular canal function but it is also a test for second mode utricular stimulation. If when the patient is tilted there is an absence of protective reaction to the tilt or if the patient tends to be thrown in the direction of the tilt, this would indicate a lesion of the vertical semicircular canals on the side to which the patient is tilted. If, on the other hand, there is a tendency for the patient to resist the tilting and to overcompensate, this would indicate a lesion of the utricles, the vertical canals being intact.

Rademaker and Garcin[55] and deKleyn and Versteegh[17] noted that if the labyrinthine lesion is of long standing it might be compensated for so that the patient might respond equally well to a tilt to either side. We have also found this to be true.

Caloric Stimulation of the Labyrinth. Vestibular tests for the semicircular canals include the rotation, the caloric and the galvanic tests but by far the most commonly used test is the caloric test. It has the advantage over the rotation test of stimulating only one labyrinth at a time. It has an advantage over the galvanic test in that it stimulates only the end organ and not the vestibular nerve as well. There has been a great deal of experimentation and discussion as to the mode of action of the caloric test and as to the best method of carrying out the test. Bárány's original explanation is the best. The temperature change sets up a convection current in the endolymph which stimulates the ampulla. If the position of the head is changed, the direction of the endolymphatic current is changed in relation to the canals. It has been shown that there are a host of factors which must be taken into consideration when stimulating the labyrinth and when observing and recording the ensuing reactions. It is generally ad-

mitted that Kobrak's method of minimal stimulation is preferable to Bárány's maximal caloric stimulation. There is some difference of opinion as to whether repeated stimulation leads to habituation and, therefore, a lessening or diminution of sensitivity. Lorente de Nó,[40] Hartmann[27] and Spiegel[58] have carried out continuous labyrinthine caloric stimulation over a period of hours on successive days without getting any marked diminution of response provided the stimulus was not excessive. It is known that the labyrinthine reaction will vary from day to day even when the method of applying the stimulus to the labyrinth is fairly constant.

Dohlman's[18] experiments have shown that a considerable amount of the temperature stimulus which is applied to the ear is required to overcome the heat resistance of the air and bone surrounding the membranous labyrinth. The labyrinth itself will respond to a change of temperature of as little as one or two tenths of a degree. Dohlman also found that the latent period of the reaction is decreased as the differences between the temperature of the stimulus and the temperature of the body is increased. The intensity of the reaction varies with the length of time the stimulus is applied to the labyrinth. The reaction of the eyes may vary depending on many factors such as whether the eyes are fixed at the time of the examination, or whether fixation is eliminated by using magnification in front of the eyes, or whether the gaze is directed towards the quick phase of the nystagmus or in the direction of the slow phase of nystagmus. Because there are so many varying factors which influence the results any method of labyrinthine stimulation which is chosen should, in so far as possible, reduce the number of variables so that results from test to test may be compared with some degree of accuracy.

Alternate Hot and Cold Caloric Stimulation. Many experimenters have used alternate hot and cold labyrinthine stimulation notably deKleyn and Versteegh,[71] Vogel,[72] Grahe,[26] Lorente de Nó[41] and Cawthorne, Fitzgerald and Hallpike.[9] The eye reactions which result from alternate hot and cold water stimulation are compared. Cawthorne, Fitzgerald and Hallpike used water at 7° C. above and 7° C. below body temperature because they believed these to be comparable stimuli. They reported that thus stimulating the horizontal semicircular canal with alternate hot and cold stimuli when it is in the vertical plane with its ampulla up (patient lying supine, face to the ceiling), the reactions which result from the ampullopetal and ampullofugal flow of endolymph are equal.

Ewald's Law. The above findings are contrary to Ewald's law that the ampullopetal flow of endolymph in the horizontal canal is a maximal stimulus, whereas the ampullofugal flow is a minimal stimulus.

De Juan[14] concluded from his experiments on rabbits that Ewald's conception of maximal and minimal stimulation of the semicircular canal varied with the position of the animal's head in space.

Lorente de Nó[37] reported from experimental (on rabbits) and clinical investigation that ampullopetal and ampullofugal stimulation of the horizontal semicircular canal are equally effective.

Grahe[26] reported that reactions to caloric stimulation of the labyrinth in patients were more definite following cold stimulation than they were following hot stimulation.

Mowrer[51] experimented with pigeons and concluded that clockwise and counterclockwise rotations were equally effective in the horizontal canal.

Working in conjunction with Stuart, Jamieson and Gaulton we carried out vestibular examinations on 100 patients with a view to testing the validity of Ewald's law. The alternate hot and cold caloric tests were done according to the method of Cawthorne, Fitzgerald and Hallpike. We were not satisfied with their assumption that water at 7° C. above and 7° C. below body temperature are comparable stimuli. The horizontal semicircular canal was stimulated with alternate hot and cold water when the patient was lying supine with the face up, so that the horizontal canal was in a vertical plane with its ampulla up. We then reversed the position of the horizontal canal in space by rotating the patient's head through 180° to the face-down position, which brought the ampulla of the horizontal semicircular canal into the ampulla-down position. This meant that whereas in the face-up position water at 7° C. above body temperature would elicit a flow of endolymph in the canal away from the ampulla, in the face-down position water at 7° C. above body temperature would elicit a flow of endolymph towards the ampulla. We could thus compare the relative values of ampullopetal flow on the one hand and ampullofugal flow on the other and we had a double check because we could compare them following the same cold stimulus on the one hand and following the same hot stimulus on the other. We found great difficulty in estimating the end point of of the nystagmus.

In the human horizontal semicircular canal (100 subjects) ampullopetal endolymphatic flow was a more effective stimulus than the ampullofugal flow in 44 per cent of cases. Ampullopetal endolymphatic flow was a less effective stimulus than ampullofugal flow in 32 per cent of cases. Ampullopetal and ampullofugal endolymphatic flow were equally effective stimuli in 24 per cent of cases. The details of this investigation are in the course of publication.

It must be remembered that in the lower animals, fishes and frogs, the semicircular canals respond to stimulation in only one direction and in each case the response is in the direction of Ewald's maximal stimulus. If there is bidirectional stimulation in birds and mammals, it is reasonable to suppose that the only direction of stimulation to which the semicircular canals of the lower animals respond is the direction of maximal stimulation in the higher animals. The reactions to stimulation of the remaining labyrinth, immediately following labyrinthectomy in man, support Ewald's ideas of maximal and minimal stimulation.

The preceding experiments show the need for further investigation before final conclusions can be drawn about the exact mode of stimulation of the human semicircular canals.

Cold Caloric Tests. The cold caloric test is used much more frequently than is the hot probably because it is difficult to arrange hot stimulation so that it will be effective and yet not be painful to the patient.

For a cold water test 2 or 3 cc. of ice water introduced by a syringe under vision and kept in contact with the ear drum for 20 seconds is a very practical ward or office test. The nystagmus, if timed from the onset of stimulation, lasts for 2 or 2½ minutes.

A simple and dependable method of carrying out the vestibular caloric test is to use a Dundas-Grant coiled tube for cold air. The air is cooled by pouring ethel chloride on the covering cloth mesh. Fifteen seconds of douching with this cold air will usually elicit a nystagmus in both the horizontal and vertical semicircular canals (head back 90° and head erect), which lasts for from 2 to 2½ minutes when the time is taken from the onset of the stimulation until the end of the nystagmus. An attempt has been made to estimate the cooling effect of this cold air and a rough estimate has shown the temperature to be reduced to about 55° or 60° F. (about 15° C.). The advantage of this method is that it is simple to carry out and can be

done in any hospital ward or doctor's office. Using the cold air makes the test safe even when there is a perforation in the drum. By this method one can test both the vertical and horizontal semicircular canals. The time required for this test is not more than ten minutes.

In deciding upon a suitable vestibular test, it is important to maintain a commonsense outlook. Crowe stated, "Too much emphasis on special examinations such as the audiometer, tuning fork and vestibular tests may lead to confusion. The diagnosis of Ménière's disease does not depend upon the demonstration of impaired hearing, shortened bone conduction or abnormal vestibular responses to the caloric or turning tests." In the differential diagnosis of central vertigo, it is important, when selecting vestibular tests, that the examination should include both the vertical and horizontal semicircular canals because in some of the intracranial lesions one set of canals may be affected and the remaining set of canals may be normal. For instance, in certain posterior fossa lesions there may be a complete absence of labyrinthine responses on one side and absence of response from the vertical canals on the opposite side while the horizontal canal on that side continues to respond.

A clinical vestibular test must be as all informative as possible and yet it should be as simple as possible. Any increased complexity in the test must give dependable information which is of clinical value before it is accepted. If we allow the test to become too complicated or if we attempt to conclude too much from a test, it will fall into disuse and disrepute.

When assessing the results or the reactions which follow a vestibular test one's attention at the present time is focused for the most part on eye reactions. The eye reactions are actually only a small part of the result of vestibular stimulation. The whole body musculature is under the influence of the labyrinth. The tests will be more revealing when our knowledge has advanced to the point where we are able to observe loss or alteration of function in muscular groups other than those connected with the eyes. Observation of past pointing of the arms following the labyrinthine stimulation is a step in this direction. The use of the tilt tests to observe general body reaction is another step but we are still a long way from the goal.

I am much indebted to Dr. E. A. Stuart for his help in searching the literature.

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XLIX

THE SYMPTOMATOLOGY OF VERTIGO

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In dealing with this part of the subject, I shall attempt the clinical approach, rather on the order of a round table discussion, depending largely upon my own experience and reading, with a minimum of specific bibliographic references. Probably Dr. McNally, a valuable contributor himself in the field of original research on the physiology of the labyrinth, has mentioned his predecessors, contemporaries and associates, and Dr. Lindsay, whose contribution to our knowledge of the pathology of the Ménière symptom complex is well known, has done likewise in his field.

My introduction to the clinical application of the discoveries of the early investigators was through the writings of Bárány; later the books of Jones and Fisher; The Handbuch der Neurologie des Ohres, edited by Hans Brunner; Brunner's lectures; and the writings of Leidler, Nylén, and of Eagleton, Lewis, Wilson, Grove, Shuster, Guggenheim and others.

A definition of vertigo is almost as difficult as is one of insanity. I have attempted to cover the subjective symptoms of disturbance of equilibrium as follows: A disturbing sensation of impaired ability to orient the position of the body in relation to surrounding objects, space or gravity. It may be merely a feeling of instability or of a seeming movement of the body or of the surroundings (rotatory in type), or of pulsion or veering of the body, or of movement of the floor, chair, ceiling or bed. Frequent accompaniments are pallor, sweat, nausea and vomiting, staggering, and sometimes clouding of consciousness. The common equivalent lay expression is dizziness or giddiness. Like headache, it may occur as a symptom of almost any ill to which flesh is heir. It is due to failure to correlate harmoniously tactile, kinetic, vestibular and optic sensations with properly coordinated efferent impulses. The fields of internal medicine, otology, neurology, and ophthalmology are all involved; that is, the origin of vertigo functional or organic, as a symptom, may lie in any of these fields, but the vertigo is manifested through disturbances or destruction of

function of the vestibular centers in the brain. The question is open as to other cerebral areas independently causing vertigo.

To facilitate the diagnosis of the causes of vertigo from the otoneurological standpoint, we attempt by observation of spontaneous phenomena and by the responses to our special tests, rotatory, caloric, postural and audiometric (including tuning forks), to determine whether the cause is functional or organic; whether a general disorder, such as toxemia, focal infection, endocrine disturbance, allergy, circulatory disturbance; or a localized lesion in the ear, the eighth nerve, the labyrinthine centers and communicating tracts; or pressure upon these structures from space-occupying lesions as a result of hypertension. I hope Dr. McNally will be able to explain the modus operandi of some of these factors.

In vertigo of sudden onset due to peripheral lesions the diagnosis is made by finding a local inflammatory process, a new growth, or trauma, associated with various degrees of hearing loss, with characteristic spontaneous nystagmus which, if present, is increased by looking in the direction of the quick component, and with the falling reaction which, if present, is influenced by the head position. In peripheral lesions of more gradual onset, as may be the case in the Ménière symptom complex, there is a history of a preceding tinnitus, a predominatingly unilateral deafness of the inner ear type, and often an imbalance in the caloric response from the two ears. (Vertigo may occur in otosclerosis.) Retrolabyrinthine and intracranial lesions must be excluded by a careful general and otoneurological examination.

More about this later, but first as to the commoner causes of vertigo not demonstrably due to a local peripheral disorder. As focal infection sources, we consider the teeth, tonsils, paranasal sinuses, gallbladder, gastro-intestinal tract, although other sources, such as the prostate gland, have been reported. A large number of toxic causes are known. The more common ones are tobacco, wood alcohol, carbon monoxide (bus passengers and employees of auto repair shops are exposed to these exhaust fumes, often combined with tetraethyl lead), and various gases occurring in industry and those used as anesthetics. To these should be added the innumerable things to which some persons are allergic. There is a long list of drugs which may produce vertigo. I want to mention the barbiturates commonly used for hypertension. It may be necessary to discontinue the drug to determine whether it or the hypertension is causing vertigo. If there are localized vascular lesions associated with the hypertension, such

as vessel spasm, thrombosis or hemorrhage, it may be possible to discover them by a special or general neurologic examination. Quinine, salicylates, chenopodium, streptomycin, the metals, such as gold, and other drugs seem to have a special affinity for the internal ear. A lessened blood supply due to anemia or failing heart action may produce what the patient describes as dizziness, especially in the upright position. I have seen this symptom in organic heart disease relieved by heart stimulation. The primary anemias, especially leukemia, should be mentioned here also. Vertigo is a common incident in practically all of the infectious fevers.

When the onset is sudden, with noise, deafness, and perhaps nausea and vomiting, or if of gradual development, it is necessary to exclude retrolabyrinthine causes: eighth nerve tumor, syphilitic neuritis, Ramsay-Hunt's syndrome, localized polioencephalitis, localized arachnoiditis, intracranial new growth, hemorrhage, or thrombosis, and such diseases as multiple sclerosis and possibly syringomyelia. Here we look for evidence of other cranial nerve involvement. In acusticus tumor it will usually be the fifth nerve which is also involved, indicated by homolateral corneal anesthesia. Jones and Fisher[6] have laid stress on the total loss of excitability of the inner ear on the affected side with loss of the rotatory component of the caloric-induced nystagmus on the opposite side as an early symptom. I doubt if most neurosurgeons would advise operation unless at least one cranial nerve other than the eighth was involved. In the apoplectic onset of hemorrhage of the posterior inferior cerebellar artery, there would be homolateral weakness and asynergia with contralateral disturbance of pain and temperature sense. Other intracranial hemorrhages and thromboses associated with vertigo may usually be identified by associated findings.

In general, otoneurologists may justly claim that they sometimes contribute materially to the answer of the question, "Is there an intracranial lesion?" and sometimes help in its localization, which may be otherwise indeterminate, and that they may be useful in discovering the presence or absence of objective evidence of trauma of the nervous system, a controversial field leading to much litigation. Grove has made notable contributions on this subject. I would like to suggest now, as I have before, that industrial surgeons would do well to consult an otoneurologist before advising patients who have had a head injury that they return to work. It would be safer for the workman and might avoid unpleasant litigation.

One of the most important objective findings, which I believe occurs only in intracranial lesions, mostly of the posterior fossa, is

postural nystagmus. If it occurs in only one direction, one must be sure that it is not merely part of a spontaneous or head movement nystagmus already present. A vertical nystagmus appearing only when the head is hung back over the table is reported by Nylén to occur principally in supratentorial lesions. Radmark[1] described his use of the tipping table to test the quick spontaneous buttressing movement of the body and limbs. He claims that of 140 cases of head injury showing no objective evidence by all other neurological tests, definite vestibular disorder was demonstrated in 60.

In the difficult discrimination between a real psychoneurosis and voluntary malingering, the exaggerated responses of the vegetative nervous system to the usual vestibular tests—pallor, sweating, tremor, nausea, vomiting, or even syncope with weak pulse—are suggestive of a real neurosis. (I have sometimes seen depressed action rather than overaction, but am not sure of its significance.) An imbalance of 25 or 30 per cent of duration time of turning or caloric nystagmus may, I believe, be taken as supporting evidence of a claim of vertigo. In hysteria I have seen convergence spasm instead of nystagmus, and twice have observed rotatory nystagmus with the head back 60 degrees and horizontal nystagmus with the head forward 30 degrees. These were both trauma cases and a central lesion was possible but not otherwise demonstrable.

Several patients with retrobulbar neuritis who were referred to me for an opinion as to paranasal sinus infection and were found negative for this, presented notable imbalance in the vertigo-nystagmus timing and the caloric and turning tests, and subsequently developed a Babinski phenomenon, loss of abdominal reflexes and spontaneous nystagmus of multiple sclerosis. Worthington[2] reports prolonged post-rotatory nystagmus as evidence of multiple sclerosis. Brunner[3] reports nystagmus on putting the head back slowly occurs in multiple sclerosis; also in cysticercosis of the fourth ventricle.

If a brain lesion is infratentorial, there is apt to be destruction of function of one or in part of both vestibular systems, that is, lack of response to stimulation by either or both cochleae and labyrinths, or a total loss of function on one side with disturbance of function on the other (as described under acoustic neuroma). Tumors of the acusticus usually begin with tinnitus and deafness and are of slow development and, as stated before, further cranial nerve involvements are usually necessary to establish the diagnosis. Other cerebellopontine angle tumors may present the same initial symptoms, but usually soon develop further evidence of intracranial pressure.

Tumors of the vermis may also present early labyrinthine disturbance, but these affect the centers first rather than the peripheral organs, and usually cause forward or backward falling. Other infratentorial lesions, unless exerting direct pressure on or otherwise affecting the vestibular centers, may not be diagnosed by the vestibular tests any earlier than by other neurological signs, but the diagnosis may be supported thereby. Medullary lesions may produce early vestibular disturbance, but are also likely to interfere with cerebrospinal fluid circulation and cause early increased intracranial pressure. Isaac Jones reported a case in which loss of the rotatory element in induced nystagmus was the only symptom beside headache. Supratentorial lesions cause vestibular symptoms by distant pressure, except in advanced cases, and show themselves by distortion of the responses rather than by destruction of function, more often by hyperirritability or imbalance of the two sides; sometimes by lessened response.

A postural nystagmus, as mentioned above, or a spontaneous vertical nystagmus is clinically quite certain to be due to a central lesion. (Vertical nystagmus has been experimentally produced in peripheral lesions in cats.[4]) I recall the case of a child having a high fever and no localizing signs or neurological symptoms, but a spontaneous vertical nystagmus. This led to examination of the spinal fluid and discovery of purulent fluid. An induced nystagmus that is not in its normal direction according to head position suggests central disturbance; so does a marked discrepancy in the duration of the induced nystagmus and the vertigo. A coarse slow nystagmus of the vestibular type, which is persistent, not influenced by head position, and increasing instead of decreasing with time, suggests a cerebellar lesion; certainly if it is toward a dead labyrinth. Dysarthria and dysphagia are late symptoms in cerebellopontine angle tumors. Inconsistent reactions, such as past-pointing in one direction with the slow component of the nystagmus in the other, is considered by Leidler[5] as evidence of cerebellar disease. In Table 1 a series of such observations by this author are quoted. The bilateral calorization test, recommended by Ruttin and Brunner, is reported by Fischer and Wolfson[7] to produce nystagmus in posterior fossa lesions but not in anterior fossa lesions. In general, one may say that a lost or diminished labyrinthine reaction may be due to peripheral disturbance, but a perverted one is of central origin.

Tumors of the midbrain may produce deafness of the inner ear type which may begin unilaterally and progress to total. This has only supporting value in diagnosis. Obviously, local treatment is

TABLE 1.

Classification of Vestibular Symptoms (after Leidler[5]).

I. Vestibular symptoms suggestive of central nervous system disease.

1. Severe attacks of dizziness over a long period with constant normal hearing.
2. Unilateral or bilateral disturbance of vestibular reaction with constant normal hearing.
3. Long lasting spontaneous nystagmus of at least second degree with normal hearing.
4. Occurrence of severe headache or increase of existing headache simultaneous with severe dizziness.
5. Perverted nystagmus reaction, especially predominence of the slow component and disturbances of the rhythm (in the direction of the conjugate deviation).
6. Postural nystagmus.
7. High-grade nystagmus without dizziness, or if both are present they are independent of each other.

II. Vestibular signs conclusive of central nervous system disease.

1. Unilateral loss of hearing and vestibular response with spontaneous nystagmus of at least second degree, to the same side.
2. Unchanged second degree nystagmus in the same direction for longer than a week.
3. Continued second degree pure rotatory nystagmus over a long period.
4. All forms of vertical nystagmus.
5. Unilateral or bilateral caloric inexcitability in the presence of normal hearing and normal rotation response.
6. One phase reactions (conjugate deviation).
7. Dizzy spells with loss of consciousness.
8. Occurrence of long-lasting changes of head posture during examination of the vestibular nerve, sometimes with somnolence or sleep.

not indicated unless associated with local disease. Any local ear condition must be carefully analyzed in the presence of suspected intracranial disease and close cooperation with the neurologist or neurosurgeon is essential, as it is with the ophthalmologist, whose knowledge of fundus findings, visual fields and eye muscle pareses may be highly contributory to the diagnosis and localization.

Perhaps it would be well to say something about the technique of the caloric test. I use 50 cc. water at 60° F. If this induces no nystagmus, I have found that neither ice water nor prolonged douching will. It is not too unpleasant. With the head back 60 degrees the onset and duration is timed with a stopwatch. The head is tipped forward 30 or more degrees to bring out the rotatory element. If one uses the minimal reaction technique, it is often necessary to repeat the test with increasing quantities of water to be sure of the presence or absence of reaction. The patient wears 16 to 20 diopter lenses and I use a head mirror for illumination. It is less awkward than the Frenzal glasses and there is nothing to get out of order.

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PATHOLOGY OF VERTIGO ARISING FROM THE PERIPHERAL VESTIBULAR APPARATUS

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The symptom of vertigo may arise from the vestibular sense organs, the primary vestibular nuclei, and probably the central vestibular pathways.

Vertigo is, therefore, a frequent symptom in many diseases of the central nervous system, particularly those involving the posterior cranial fossa, as well as in disease of the peripheral sense organ.

This discussion is concerned with the pathology which gives rise to vertigo from the peripheral vestibular mechanism which includes the sense organ and the vestibular nerve. The localization of a lesion to the peripheral apparatus is based clinically upon a combination of auditory and vestibular symptoms.

The clinical localization in the case of vertigo of central origin is usually based upon the associated symptoms and signs arising from the central nervous system.

In everyday clinical experience, however, the majority of cases of vertigo are not associated with auditory symptoms or with other signs of disease of the central nervous system. They are usually classed under the heading, pseudo-Ménière's syndrome.

Any discussion of the pathology of vertigo must take this group into consideration since here the differentiation of a central from a peripheral origin usually cannot be positively established.

The usual occurrence of vertigo in such diseases as cerebellar abscess, in tumors located in the posterior fossa, and in degenerative disease such as multiple sclerosis has been explained by the demonstrable involvement of the vestibular nuclei and tracts by pressure or degeneration. Vertigo may apparently be caused by involvement of the central vestibular apparatus which may be of a reversible nature and not easily demonstrated histologically. The observation of

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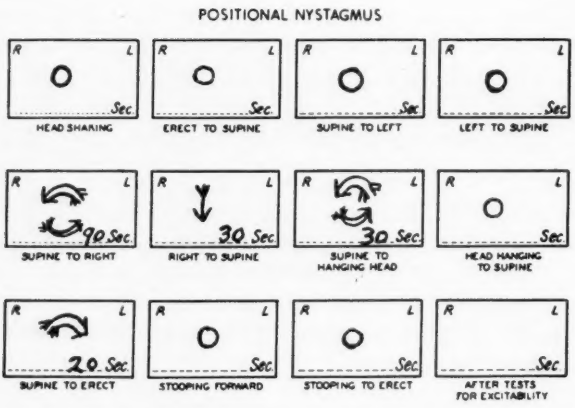


Fig. 1, Case 1.—M. M. Record of postural tests made nine days after falling and striking the back of his head on the ice. There were no other postconcussion symptoms, no auditory symptoms, and no spontaneous nystagmus before doing postural tests. The rectangles are arranged in the order in which the postural tests were carried out.

Nylen[1] that in posterior fossa tumors a vertigo occurring in all positions of the head was present in 60 per cent while a postural vertigo was present in 90 per cent appears to be characteristic for other central lesions[2] in the posterior fossa.

For example, the concussion injury which results from a blow on the head is frequently followed by vertigo. In relatively mild injuries the vertigo may be the only disturbing symptom but may last for several weeks and is characteristically of the postural type (Fig. 1).

Case 1.—A 57-year-old male fell on the ice while skating, striking the back of his head with sufficient force to cause a small laceration of the scalp. There was no loss of consciousness. He continued skating but within a short time became aware of dizziness after movement. The next morning he noted dizziness when he assumed certain positions. Headache was absent. He was seen nine days later with the same complaint of dizziness when he assumed certain positions. There was no vomiting.

The general examination was entirely negative.

There was no deafness or tinnitus at any time.

There was no spontaneous nystagmus in the upright position. Postural tests produced vertigo with positional nystagmus in the right lateral position, in the supine position when assumed from the right lateral, in the head-hanging position, and upon sitting up from the supine position.

The positional nystagmus altered in direction depending upon the position thereby corresponding to Type 1[3] (Nylen). (See Fig. 1.)

Histologic changes have been demonstrated in the vestibular nuclei of animals following concussion injuries.[4]

When clinical evidence may exist in the form of a positional nystagmus which reverses its direction, a spontaneous vertical nystagmus, or a perverted response to caloric stimulation that would indicate a central localization for some cases of pseudo-Ménière's syndrome, histopathologic evidence has not been obtainable and proof is therefore lacking. Examples are the vertigo, usually postural, which occurs with some acute infections of the respiratory tract, with arteriosclerosis and hypertension, in some cases with hypotension, vasomotor instability, and in intoxications due to certain drugs, of which the most recent example may be the severe vertigo without auditory symptoms but with bilateral loss of cold caloric response which occurs in some cases after streptomycin therapy. In these conditions the occurrence of a postural vertigo and positional nystagmus which changes direction with a change of position of the head may prove to be a fairly reliable indicator of a centrally located lesion. In the case of known lesions this type of positional nystagmus has been observed to be indicative of a central localization,[1, 2, 5] whereas a positional nystagmus of constant direction has been found in both peripheral and central lesions.

Vertigo of Peripheral Origin. The most common cause for vertigo of peripheral origin in years prior to chemotherapy and antibiotics has probably been labyrinthitis of the diffuse type.

The pathways by which the labyrinth may be invaded by an inflammatory process have been demonstrated by the study of serial sections of the temporal bones. These have been outlined in various standard texts and need not be reviewed.

A characteristic feature of diffuse labyrinthitis of either otitic or meningeal origin is that invasion of the perilymphatic spaces occurs primarily. In mild cases which have been arrested in the "serous" stage various degrees of fibrous connective tissue proliferation and some osteogenesis are found in the perilymphatic spaces. The endolymphatic system may show no signs of invasion, but some degree of hydrops is usually found.

While little evidence of inflammatory reaction may be visible within the endolymphatic system, the occurrence of hydrops is usually associated with degeneration of the organ of Corti and the

Fig. 2.—F. W., male, aged 12. Photograph of the base of the skull showing a large meningioma occupying the cerebellopontine angle on one side. The tentorium has been removed along with part of the dura from the middle fossa on the side of the tumor to show the extension through the petrous ridge. There was no demonstrable auditory or caloric response from the affected side.

stria vascularis. Advanced degeneration of the spiral ganglion and nerve fibers is usually found as well. Degenerative changes in the vestibular sense organs may not be as susceptible to histologic demonstration. Irreversible changes probably occur less readily but are demonstrable in advanced cases.

The term "post-hydropische degeneration" was applied by Wittmaack to the degeneration occurring in serous labyrinthitis. It is sometimes observed, however, that the degree of degeneration of ganglion cells and nerve fibers has occurred out of proportion to the degree of hydrops, or even before a hydrops has developed. This suggests that the degeneration of the ganglion and nerves may be in the nature of a toxic process occurring independently of the

hydrops. Rapid degeneration of the organ of Corti and of the stria vascularis appears to be characteristic of a hydrops which has resulted from an inflammatory reaction in the perilymphatic spaces. This would suggest a degeneration due to altered chemical properties of the fluid at the time of occurrence of the hydrops.

Degeneration of the vestibular sensory apparatus is less easily demonstrated histologically. In the presence of labyrinthine irritation the vestibular apparatus gives symptoms along with the cochlear apparatus and appears equally sensitive, but it appears to have a greater capacity for recovery.

Circumscribed Labyrinthitis. Circumscribed labyrinthitis is diagnosed by the presence of the fistula symptom.

It occurs in cholesteatoma most often and usually involves the horizontal semicircular canal. It has been observed also to involve the superior semicircular canal, producing a vertical nystagmus.[6]

The fistula symptom is common in congenital syphilis and is due to erosion of the bony capsule producing a soft area in the wall of a semicircular canal, thereby allowing fluid movement in that canal upon exertion of static pressure on the drum and conducting mechanism[7] or the windows.

A circumscribed labyrinthitis has also been observed in tumors of the temporal bone and in granuloma involving the middle ear spaces. The symptom occurs when the bony capsule is eroded through. If the erosion exposes the membranous structure of a semicircular canal, the situation is created for production of the fistula symptom.

Tumors. Vertigo of peripheral origin may be caused by erosion of the wall of the labyrinth by tumors involving the temporal bone. It also occurs by compression and invasion of the vestibular nerve in the case of tumors arising in the internal auditory meatus, such as acoustic neurinoma, von Recklinghausen's neurofibromatosis, or extension of tumors of the meninges or the brain into the meatus.[8]

Tumors in the cerebellopontine angle involve the eighth nerve, either primarily as in the case of acoustic neurinoma or by pressure, for example in meningioma (Fig. 2). While the tumor in the meatus or in the angle usually produces a profound loss of both auditory and vestibular function on the affected side primarily, the type of nystagmus which occurs in late stages is not typical for loss

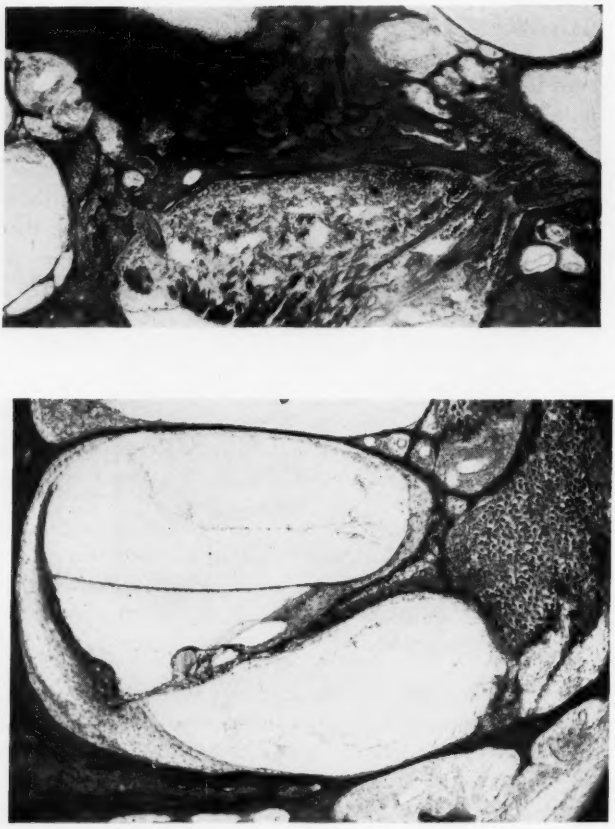


Fig. 3.—Photomicrographs of a monkey's ear a few minutes after a blow to the skull. The animal was perfused before fixation; therefore the vessels are empty. In Fig. 3*a* can be seen extravasated blood cells in the subarachnoid space extending along the vessels and nerves in the porus acusticus.

In Fig. 3*b*, free blood cells are seen in the connective tissue of the modiolus. Strands of fibrin and red blood cells are seen in the perilymphatic spaces.

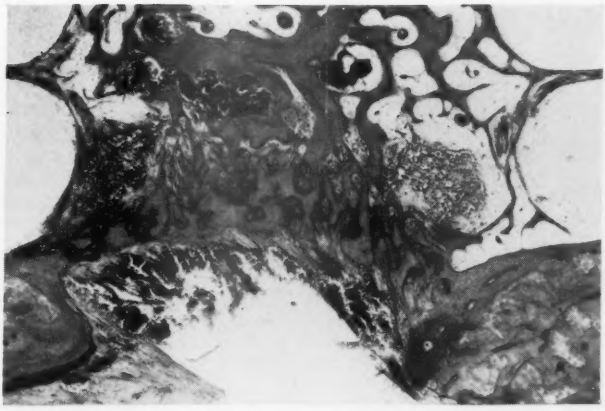


Fig. 4.—Photomicrograph of the modiolus of one cochlea in a case of subdural hematoma, without fracture. Collections of red blood cells are seen between the nerve fibers in the internal meatus, in the porus acusticus, the spiral ganglion and the loose connective tissue around the blood vessels.

of one labyrinth. Instead of a nystagmus with constant direction it may occur in the direction of gaze, sometimes called a fixation nystagmus, and is the result of pressure or displacement on the central nervous system as well as the loss of one labyrinth.

Blows to the skull in man have produced a temporary threshold loss of hearing at the C_5 frequency without vertigo.[9] This has been interpreted as the same type of acoustic trauma as is caused by the shock pulse travelling through the air. The severe deafness, tinnitus and vertigo which sometimes follow a blow to the head, without demonstrable fracture, are attributed to concussion of the ear. Because the symptoms do not appear immediately but may develop over a period of hours, the etiology has been considered as hemorrhage within the labyrinth.

Histopathologic proof has not been obtained in man.

Experimental blows to the skulls of animals produce subarachnoid hemorrhages and extravasation of blood in the tissues surrounding nerve fibers and blood vessels in the porus acusticus (Fig. 3). Small hemorrhages and degeneration of cells in the cochlear and vestibular nuclei have also been described.[10] Blood cells may be ex-

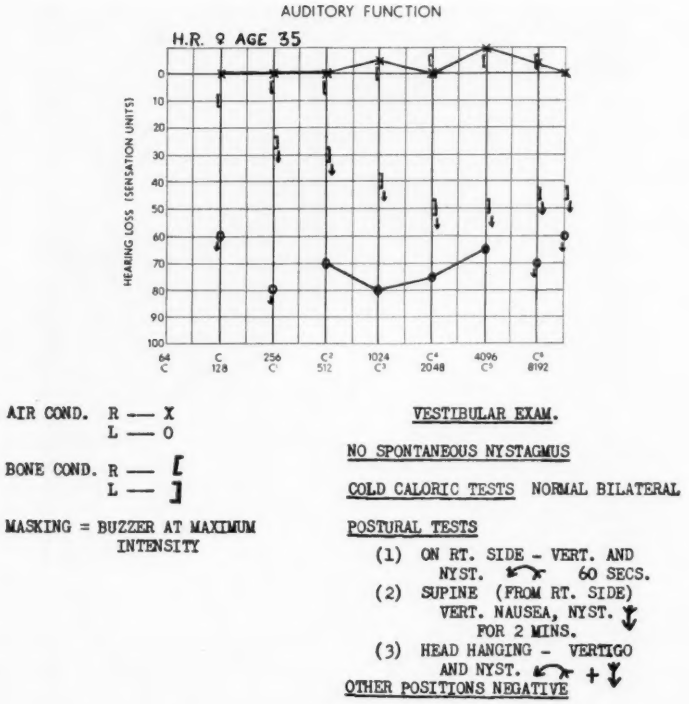


Fig. 5, Case 2.—Apoplectic onset of Ménière's syndrome three years earlier in a previously healthy ear. Postural vertigo, tinnitus and profound unilateral deafness persist. The chart shows the auditory pure tone thresholds for air and bone conduction and the results of the vestibular examination.

The positional nystagmus varied in direction depending upon the position but did not reverse.

travasated in the connective tissue meshwork supporting the vestibular sense organs and also lie free in the perilymphatic spaces. The question as to how much extravasation of blood cells is necessary to produce interference with function seems not to have been answered.

Subarachnoid hemorrhage can also be demonstrated in man with severe head injuries in the absence of fracture (Fig. 4). Subarachnoid hemorrhage is commonly found at the porus acusticus after craniotomy[11] and may extend along the nerve fibers and vessels in the

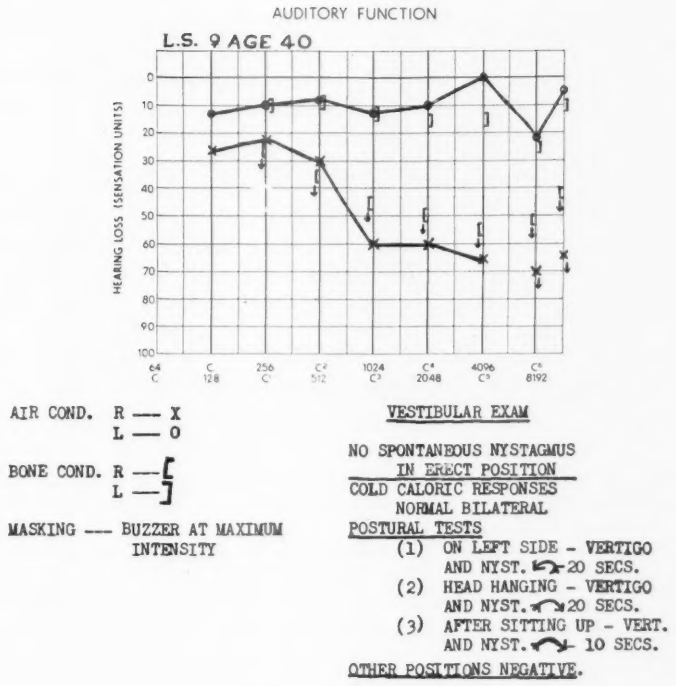


Fig. 6, Case 3.—Sudden onset of noises, impaired hearing and gradual onset of vertigo. The chart shows auditory thresholds for pure tones by air and bone conduction and the results of the vestibular examination one year after the onset. The positional nystagmus in this instance corresponds to Type 1 (after Nylen) in that the direction reversed upon certain changes of position. This type is seldom seen in a peripheral vestibular lesion.

modiolus. Patients observed by us who have recovered from craniotomy usually do not show any hearing defect or vertigo however.

Extravasation of blood along the nerve fibers and vessels appears insufficient, therefore, to account for a loss of function. The profound loss which sometimes occurs suggests a frank hemorrhage into the inner ear spaces.

The vertigo which follows cerebral concussion without deafness appears to be explainable by the alterations in the central vestibular nuclei.[4, 9]

Hemorrhage and Thrombosis. Since a vascular accident in the labyrinth is not a fatal disease, histopathologic material is difficult to obtain. Pathologic evidence is limited to cases of leukemia in which a serofibrinous labyrinthitis has been found on postmortem examination. This has been attributed to hemorrhage into the perilymphatic space.[12]

The clinical diagnosis of a vascular accident is made in the case of the sudden or apoplectic onset of deafness, tinnitus and vertigo in a previously healthy ear which leaves a permanent impairment of function. The degree of impairment may apparently vary from a moderate nerve deafness without vertigo in the mildest cases, to a more marked deafness with vertigo, which may persist for years as a postural symptom only, or a more profound and permanent loss of both auditory and vestibular function (Figs. 5 and 6).

CASE 2.—H. R., female, aged 36, complained of deafness and noises in the left ear and dizziness. The symptoms came on suddenly three years earlier.

Vertigo was accompanied by vomiting at the onset. After about two weeks the dizziness had receded in severity and occurred only in certain positions. There were no further attacks but the postural vertigo has persisted without further improvement until the present. It is noted particularly on lying on the left side. A buzzing noise in the affected ear and a sense of pressure or blockage varying in severity from time to time has been troublesome. Her hearing is greatly depressed.

There were no ear symptoms previous to the onset of the present complaints.

The general examination was essentially negative.

Local ear, nose and throat examination showed normal tympanic membranes. The right maxillary sinus was filled by a large polyp projecting into the nasal passage. There was no evidence of suppuration.

The auditory examination revealed profound nerve deafness on the left side but some residual hearing was present with adequate masking. There was normal hearing on the right.

There was no spontaneous nystagmus in the upright position. The fistula test was negative. There was a normal cold caloric response in both ears. In the postural tests vertigo was accompanied by positional nystagmus in the supine and head-hanging positions (Fig. 5).

A radical antrum operation on the right revealed the antrum to be filled by polypoid and cystic tissue.

Nine months later the complaints referable to the left ear remained unaltered.

CASE 3.—L. S., female, aged 40, experienced a sudden onset on March 1, 1946, of screaming and whistling noises in the right ear, associated with deafness and a gradual onset of vertigo which reached a maximum about one week later. The vertigo has been postural, brought on especially by placing her head backwards, lying on one side, and after sitting up. It has not decreased in the period of one year since the onset. She is unable to tolerate high-pitched loud sounds or music. She has had no headaches. Her ears were normal previous to this episode. There has been no appreciable change in the symptoms during the past year.

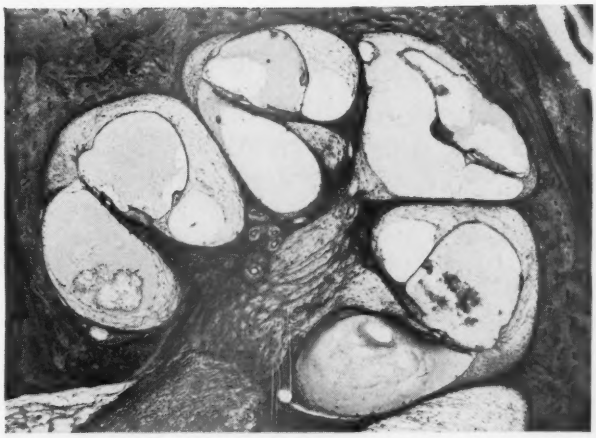


Fig. 7.—Photomicrograph of a section through a rabbit's cochlea about one month after making an opening through the capsule into the apical coil. The bone has healed; a serous labyrinthitis has occurred with diffuse fibrosis involving mainly the vestibular scala. The spiral ganglion and the associated nerve fibers show a marked degree of degeneration. The cochlear duct is dilated. The endolymph shows an abnormal staining reaction and degenerative changes of the stria vascularis, tectorial membrane and Corti's organ are evident.

The general examination, including the neurological, was essentially normal.

The nose and throat examination was negative. The tympanic membranes were normal. There was moderate high-tone nerve deafness in the right ear (Fig. 6).

No spontaneous nystagmus was present in the upright position. The fistula test was negative. There was a normal cold caloric response in each ear. The postural tests revealed marked vertigo and nystagmus in the head-hanging position, on the left side, and on sitting up. The positional nystagmus was counterclockwise in the left lateral position but clockwise in the head-hanging position and after sitting up quickly.

Comment: The possibility of a thrombosis due to an arteriosclerotic lesion affecting only the cochlear division of the arterial supply has been suggested to explain sudden deafness on the basis of vascular distribution to the labyrinth.

Experimental interruption of arterial supply by crushing the internal auditory artery in the meatus[13] has shown varying degrees

of degeneration of the neural elements as well as proliferation in the labyrinthine spaces. The injury could not be limited to the vascular supply alone, however, by this experimental method.

While a vascular accident would offer the best explanation for the apoplectic type of a Ménière's syndrome occurring as a single episode in a previously healthy ear after middle age, factual information regarding the pathology is at present inadequate. A toxic neuritis sometimes cannot be differentiated.

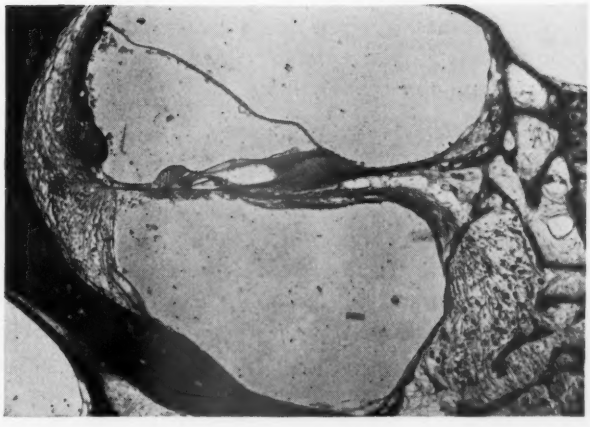
Toxic Neuritis or Neurolabyrinthitis. Impaired hearing and vertigo may be caused by a toxic disturbance of the inner ear. When these two symptoms occur together, the auditory function apparently is the more susceptible to injury and to irreversible damage.

The pathology in the inner ear in deafness following mumps or in a so-called Ramsey-Hunt syndrome has not yet been demonstrated. Experimentally, however, some information has become available.

Degeneration of neural elements (Corti's organ, spiral ganglion and nerve fibers) has been produced in animals by placing common salt and other electrolytes against the round window membrane;[14] also by using toxins in a similar manner.

Degeneration of the peripheral cochlear neuron occurs in serous labyrinthitis. This has been frequently demonstrated in man after infections and in some animals following experimental injury to the wall of the cochlea (Fig. 7). In the latter the presence of infection could not be disproven. The inflammatory changes are primary in the perilymphatic spaces in such cases. A hydrops of the endolymphatic spaces is also found. In hydrops of this type, occurring as a result of inflammatory changes, it is common to see an extensive degeneration of both Corti's organ and the stria vascularis. The placing of electrolytes[14] on the round window membrane has produced similar degenerative change.

Recent experiments upon fenestration in animals have demonstrated varying degrees of degeneration of the peripheral cochlear neuron. These experiments have provided some evidence that degeneration may affect the peripheral cochlear neuron alone or, as it becomes more marked, may be associated with a hydrops (Fig. 8). A still more advanced stage is represented by a hydrops and a pro-



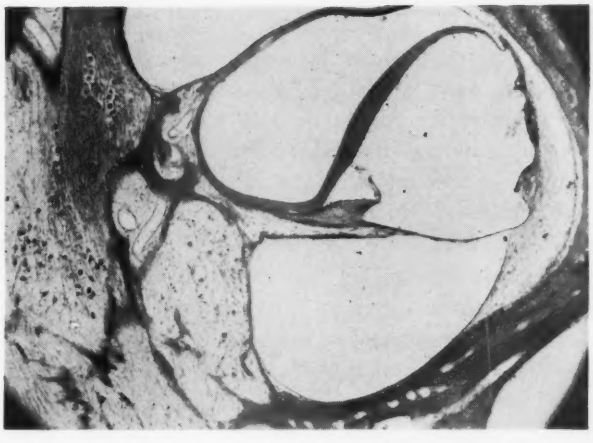


Fig. 8*a*.—Photomicrograph of a section through the middle coil of a monkey 14 months after fenestration, with a patent fenestram. Moderate degeneration of the spiral ganglion and nerve fibers has occurred (Compare with Fig. 4). The cochlear duct and its contents appeared normal.

Fig. 8*b*.—A photomicrograph of a section through the middle coil of another monkey, five months after fenestration, which shows advanced degeneration of the peripheral cochlear neuron, a slight degree of hydrops, complete degeneration of Corti's organ and partial degeneration of the stria vascularis. Some alteration of composition of perilymphatic fluid is indicated by the deeply staining coagulum overlying Reissner's membrane.

liferation of connective tissue in the perilymphatic spaces. The hydrops in this instance is accompanied by degeneration of both Corti's organ and the stria vascularis. This suggests that a change in chemical constitution of the endolymph may be the basis for both the hydrops and the degeneration of structures within the cochlear duct.

The histopathologic changes in the vestibular sense organs and nerves have been less evident but are also less easily demonstrable by histologic methods than in the case of degeneration of the cochlear neuron or Corti's organ. Degeneration of the sense organs occurs in association with the hydrops accompanying inflammatory changes in the perilymphatic space.

Idiopathic Hydrops of the Labyrinth or Ménière's Disease. So-called idiopathic hydrops of the labyrinth differs from the hydrops occurring as the result of serous labyrinthitis. The perilymphatic spaces are usually free from inflammatory changes. The cochlear aqueduct is open. Corti's organ and the stria vascularis do not show the marked degenerative changes seen in serous labyrinthitis. The peripheral cochlear neuron may exhibit little or no histologic evidence of degeneration, even in advanced cases.[15, 16] In some there may be degenerative changes in the vestibular sense organs in the late stages.[17] The most common lesions to be seen histologically which might explain vertigo are the varying degrees of distortion of the membranous structures in the regions of the junctions of the ampullae with the utricle. The suggestion has been made[17] that these might interfere with normal function of the cupula in the ampulla, thereby disturbing equilibrium.

Such an explanation might fit in with the clinical experience that the vertigo is extremely variable in frequency, duration and severity. The vertigo may or may not seem to have a direct relation to the fluctuations in auditory acuity, sense of blockage in the ear or noises or it may never exist as a complaint.

The development of this type of hydrops has not yet been adequately explained. The comparative absence of neural degeneration suggests an increased secretion of normal endolymph rather than any alteration of chemical constituents. It offers a contrast to the hydrops which occurs in inflammatory conditions.

SUMMARY

1. The pathologic disturbances which are associated with most clinical conditions giving origin to pseudo-Ménière's syndrome (vertigo without auditory symptoms or central neurologic signs) are not known. The localization has rarely been proven. The postural vertigo which is a characteristic of this group usually corresponds to the type which is most frequent in proven disease of the posterior fossa in that a positional nystagmus occurs which changes direction with alteration of the position of the head. A central origin is therefore suggested.

2. Vertigo of peripheral origin has been satisfactorily explained in labyrinthitis of various types, tumors and fractures of the labyrinth.

3. In concussion of the labyrinth the pathologic lesion has not been adequately demonstrated. A temporary threshold dip at the C_5 frequency has been shown. A subarachnoid hemorrhage which extends along the vessels and nerves into the porus acusticus and even into the perilymphatic spaces is common after head trauma and following craniotomy.

The degree of hemorrhage into the porus acusticus and the perilymphatic space which is necessary to interfere with function is, however, indefinite. Probably a frank hemorrhage into the labyrinthine spaces is necessary for any marked disturbance of function.

The pathologic explanation for the apoplectic onset of deafness, tinnitus and vertigo in a previously healthy ear has not been demonstrated. An interruption of circulation or a hemorrhage are considered to be most probable.

4. The pathogenesis of a toxic neuritis or neurolabyrinthitis is not clear in all cases. For example, the occurrence of progressive neural degeneration in otosclerosis, as well as that which sometimes occurs as a complication after the fenestration operation, has created special interest.

5. The histopathologic disturbance in idiopathic labyrinthine dropsy lacks an etiologic explanation.

Degeneration of neural elements appears to be a late accompaniment of this type of hydrops, whereas in serous labyrinthitis the degeneration of neural elements occurs early.

Degeneration of sensory structures within the endolymphatic spaces and the stria vascularis appears to parallel the development of hydrops in serous labyrinthitis but may be slight or absent in comparatively long-standing cases of idiopathic hydrops

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LI

INCIDENCE OF VERTIGO IN NEUROLOGICAL CONDITIONS

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There is general agreement that true vertigo should be limited to the subjective sensation of rotation either of the individual or of his environment. The patient generally uses the term dizziness to describe this sensation. In its minimal manifestations there may be a subjective sensation resembling giddiness. These mild degrees must be differentiated from giddiness produced by alterations in cerebral blood flow incident to postural change, carotid sinus reflexes, and vagal syncope, and also from phobic reactions with accompanying anxiety.

The neurological evaluation of vertigo is dependent upon the paraphenomena associated with it. These vary with the location and the etiology of the lesion, and since for awareness of vertigo to exist the cerebral cortex must be activated, both brain stem and cortical levels of function must be examined whenever it is suspected that the source of the vertigo is not peripheral. The manner of development of the symptom, the distribution of associated symptoms and signs, the laboratory, and x-ray findings lead to the proper interpretation. In general, lesions of acute onset, such as vascular accidents, are more apt to produce vertigo than insidiously developing lesions, as for example, infiltrating tumors.

General physiological principles that apply to other sensory systems are equally useful in understanding the neural basis of vertigo. As is the case with other proprioceptive fibers the vestibular may be presumed to be continuously active and the sensory inflow continuously adjusted for balance between the two sides. Contemplation of the central basis for maintaining continuous activity and shunting it into the reflexogenic and cortical relay paths re-

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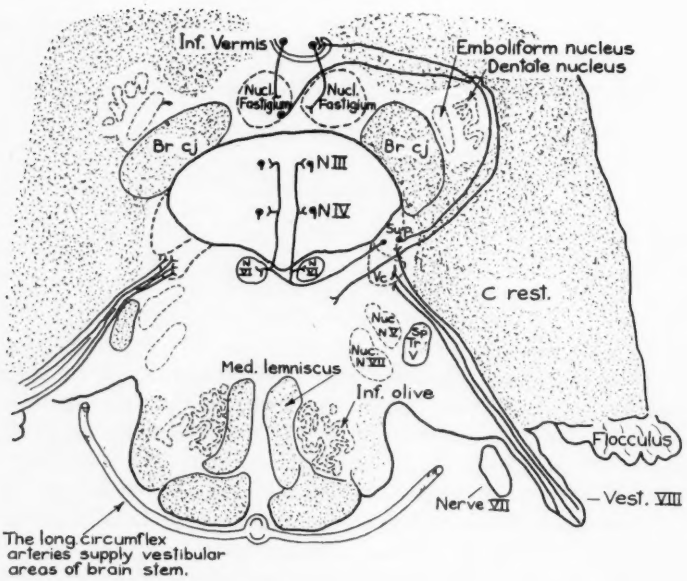

Fig. 1.—Diagram of a transverse section through the brain stem at the cerebellopontine angle indicating the tracts and nuclei encountered by the vestibular division of the VIIIth nerve as it passes obliquely between the restiform body and the spinal tract of the trigeminal to reach the floor of Ventricle IV. The principal connections of the vestibular nuclei with the cerebellum and with the medial longitudinal fasciculus are indicated. Principal abbreviations are: Vestibular nuclei—*VC*, vestibulo-cerebellar; *Sup*, superior. *Nuc N VII*, motor facial nerve nucleus; *Nuc N V* and *Sp Tr V*, nucleus and spinal tract of trigeminal nerve respectively; *C rest*, restiform body. The long circumflex artery which gives branches to supply the vestibular nerve entry territory is the anterior inferior cerebellar. More caudally, the posterior inferior cerebellar, another long circumflex, supplies a like territory.

quires a more complicated concept of the brain stem vestibular mechanism than is drawn conventionally for the paths between vestibular nuclei, eye muscle nuclei and cerebellum (Fig. 1). Overly simplified diagrams do not take into account the reverberating circuits postulated by Lorente de Nó[1] for prolonging or augmenting vestibular effects. A reverberating mechanism has been shown by this author to necessitate the interaction of nerve cells of the brain stem reticular substance. A diagram modified from Lorente de Nó is presented in Fig. 2. It may be presumed that the median longi-

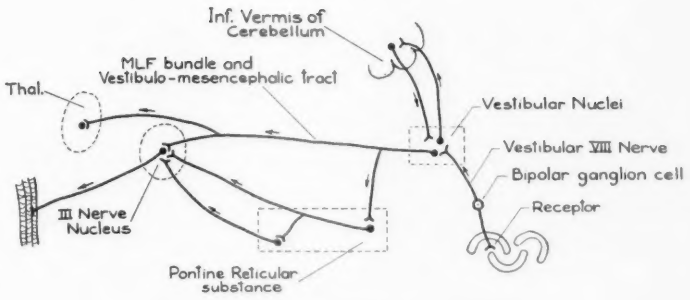

Fig. 2.—The principal vestibular connections with the brain stem, cerebellum and thalamus are indicated schematically. The delay circuits through the pontine reticular substance, emphasized by Lorente de Nó, play an important role in the vestibular mechanism.

tudinal fasciculus and vestibulo-mesencephalic tracts in addition to providing links in the reflexogenic vestibular paths to the eye muscle nuclei also establish connections with the cortical level through the thalamus.

Thus vertiginous symptoms might arise in one of three ways: preponderant activity arising from the vestibular receptors of one side, diminution of activity arriving from the other side, or gradual or sudden alterations in the level of excitability of the centers. Such changes in level of excitability might be expected to occur as lesions develop in or adjacent to the vestibular nuclei or paths, or affect the activity of internuncial chains through the reticular substance. Alterations in the level of cortical excitability without peripheral or brain stem disturbance can also cause vertigo.

In Table 1 we present a systematic classification of vertiginous conditions based upon the prevalent pathology that produces vertigo when the vestibular division of the auditory nerve is affected intra-aurally or in its course through the cerebellopontine angle, and when the vestibular centers are affected in the brain stem or at the cortical level. Case reports will be used to illustrate the procedure in diagnosis and treatment in those conditions frequently overlooked as causes of vertigo.

TABLE 1.

COMMON SITES OF DISTRIBUTION
OF VERTIGINOUS LESIONS.

End Organ and Petrous Course of Vestibular Nerve	Hydrops of endolymphatic sac Related hypothyroidism Degeneration of vestibular division, VIIIth nerve Unknown etiology Head injury
Vestibular Nerve in Cerebellopontine Angle	Acoustic neurinoma Unusual but occurs Tumors adjoining vestibular division, VIIIth nerve Meningiomas most likely Vascular conditions Aneurism Anomalous course of vessels and arterial loops Arteriosclerotic vessels, pressure of Infections Pyogenic abscesses Granulomata Lues Torula Platybasia Arachnoiditis
Brain Stem and Cerebellum	Vascular conditions, particularly affecting vestibular nuclei Thrombosis or hemorrhage Posterior-inferior cerebellar Anterior-inferior cerebellar Multiple sclerosis Plaques occurring in pontine reticular substance Encephalitis and encephalomyelitis Tumors Pontine Cerebellar, particularly when inferior vermis is involved
Cerebral Cortex	Epilepsy Vertiginous Aura of Migraine Related hypothyroidism Tumors
Functional Disorders	Anxiety hysteria with nucleus of vertigo
Drugs and Toxic Conditions	Toxic Alcoholism Dilantin Tridione

CASE 1.—*Platybasia.* A 73-year-old white male was examined for the first time by us on November 8, 1946. At that time we obtained a history of dizziness with difficulty in walking and right-sided headache for a period of 15 years. During the previous five years these symptoms had so increased in severity that the patient was barely able to get about. Dizziness was made worse by turning the head to either ide. His old hospital records revealed that in 1923 he presented signs and symptoms referable to the vestibular system. There was a complaint of dizziness at that time, together with nystagmus on lateral deviation. At that time the suggestion was made that the patient might have a cerebellar tumor.

On the last admission, in 1946, the patient had an unsteady gait with a wide base. There was a downward drift of the right arm on posture holding. The deep reflexes of the right upper extremity were slightly increased. The abdominal reflexes were equal and active. The patellar and ankle reflexes were absent. There were no pathological toe signs. Vibratory and position senses were markedly diminished in both lower extremities. There was slight diminution of the same modalities in the fingers. The pupils were equal, round, and reacted well to both light and convergence.

There was a marked nystagmus on lateral deviation of the eyes, more to the left than to the right. The left ear was completely deaf. The Weber test was referred to the right. The Rinne test was positive on the right. Caloric reactions with water at 68° F. revealed a dead labyrinth on the left. On the right side a sense of vertigo was produced in about 23 seconds with neutralization of the nystagmus obtained on right lateral deviation. Audiograms showed deafness on the left and marked diminution in hearing on the right, particularly above 2048 vibrations per second. The right motor fifth nerve showed suggestive weakness.

His blood pressure was 180/105, but varied downward to 150/90. The pulse was 80 per minute. The electrocardiogram showed a left axis deviation. Gastric analysis revealed: free acid 0, total acid 16. The blood tests showed the following: blood sugar 96 mg. per 100 cc., nonprotein nitrogen 24 mg. per 100 cc., diastase 146 mg. per 100 cc. per hour, white blood count 7,500 cells per cubic mm., red blood count 4,100,000, hemoglobin 12.6 grams. The stool examination was negative; urinanalysis negative. Spinal fluid showed normal pressure; protein 71 mg. per 100 cc.; Kahn negative; cells 2; colloidal gold curve 0000000000. X-ray films of the skull revealed a scaphoid shape. The basal angle was markedly increased to about 160°. The tip of the odontoid process was well above Chamberlain's line. The x-ray diagnosis was platybasia (Fig. 3).

Comment: In the last year four cases of platybasia have come to our attention having as the presenting symptom chronic vertigo, paroxysmal or continuous. Basilar impression is similar to platybasia in the manner of production of symptoms and is characterized by invagination of the base of the skull at the foramen magnum. The basal angle is also increased in basilar impression. The difference between the two is that in platybasia we usually see a shallow posterior fossa. In basilar impression the latter is apt to be deep. Basilar impression may be either congenital or acquired. Paget's disease is the most common cause of the acquired form. Accompanying the change in the basal angle in both conditions, the petrous pyramids are usually rotated forward. There is also a disruption of the normal anatomical relationship between the clivus, the brain stem, and the

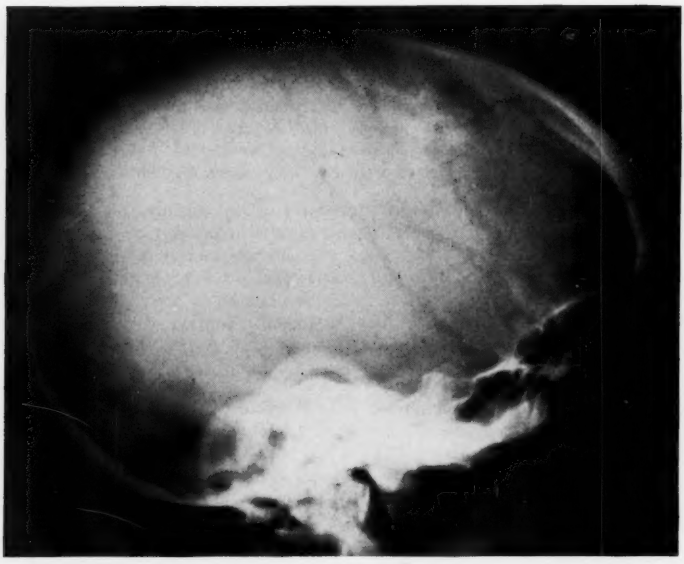

Fig. 3, Case 1.—A lateral view of the skull in a case of platybasia having a long history of vertigo. The chief diagnostic feature of platybasia roentgenographically is the projection of the tip of the odontoid process above Chamberlain's line. There is an increase in the basal angle with rotation of the petrous pyramids.

structures occupying the posterior fossa. At operation arachnoidal bands are frequently found to be constricting the brain stem and cerebellum. The treatment is surgical decompression. The usual clinical picture of both basilar impression and platybasia simulates multiple sclerosis, syringomyelia, cerebellar tumor, or hydrocephalus. It is interesting to note in this patient that the neurologist who examined him 23 years ago suggested, on the basis of ataxia and nystagmus, that the patient might have a cerebellar tumor. We wish to point out that vertigo can be the outstanding symptom in this type of condition. When the symptom is chronic and there is evidence of involvement of the brain stem, platybasia should be suspected.

Case 2.—*Meningioma of the Cerebellopontine Angle.* A 35-year-old, married female was admitted to the hospital on December 3, 1941, with a history of "dizzy spells" of minimal degree for somewhat longer than five years. During the year preceding admission these episodes increased in severity. Sudden changes in posture, such as standing or turning the head, would cause staggering. During

Fig. 4, Case 2.—Towne's view of the petrous pyramids showing a small area of calcification representing the site of a meningioma attached to the vestibular nerve in the cerebellopontine angle. The patient gave a history of intermittent vertigo that was relieved by removal of the meningioma.

the year previous to admission she also began to notice a humming sound in her left ear and shortly afterward was aware of diminished hearing on that side. Three weeks prior to admission she suddenly lost consciousness while standing in her kitchen. She later described this by saying: "The linoleum came flying up in my face." For two weeks before admission she had noticed double vision on left lateral gaze and some difficulty in using her left hand with its usual dexterity.

Physical examination revealed a small hyperkinetic woman whose gait became unsteady on rapid turning. When she walked with eyes closed, there was a tendency to veer to the left. In the Romberg position she fell to the right. She was unable to stand on one foot. There was some flattening of the left nasolabial fold. The deep reflexes were equal and active. There were no pathological toe signs. Ataxia was observed on heel to knee test on the left. Past-pointing to the left was also noted. The eye grounds were normal. No nystagmus was observed. Audiograms revealed depression of all tones on the left. Caloric reactions, with water at 68° F., indicated a dead labyrinth on the left and a response from the horizontal canals only on the right. With ice water a slight reaction was obtained from both sides. Radiograms revealed an area of abnormal density projecting just above the mesial part of the petrous ridge on the left side in the Towne position (Fig. 4).

At operation a small meningioma was found at the point of entrance of the eighth nerve into the brain stem and ventral to the restiform body. This was found to be attached to the sheath of the eighth nerve although the latter was not involved in the tumor. After a rather difficult postoperative course, the patient made a very satisfactory recovery and on last examination was symptom free.

Comment: Tumors in the cerebellopontine angle may cause vertigo. While this symptom does occur in acoustic neurinomas, it is unusual. Vertigo is more apt to be a symptom of tumors not arising from the vestibular division of the eighth nerve. In this patient dizziness and deviation to the left were the earliest and outstanding symptoms. This lack of the usual orderly sequence of events as seen in acoustic neurinomas serves for differential diagnosis. In our experience oblique stereoscopic films frequently serve to reveal abnormal calcification in the cerebellopontine angle.

The vagal reflexes arising from stimulation of the vestibular system may result in loss of consciousness. The latter may overshadow the symptoms of vertigo. Only a careful history will serve to elucidate the relationship between the two symptoms.

CASE 3.—*Aneurysm of a Long Circumferential Artery.* A 27-year-old, white female was first admitted to the Neurosurgical Service of Barnes Hospital on October 13, 1946. Her history revealed that for a period of three years she had had five or six episodes of vertigo, weakness, headache, and fluttering of her heart. The attacks would commence suddenly but were sufficiently severe to incapacitate her from work for a period of three to four months on each occasion. She was treated for sinusitis, anemia, and heart trouble. Seven weeks before her first admission she had sudden onset of severe occipital headache during the night. The next morning she staggered markedly, tending to veer to the left. Her vision became blurred. She was nauseated and vomited. The latter symptoms lasted for two days. After a week the vertigo began to improve but she was still unable to walk without support. Eleven days after the onset she awoke from a nap with a loud roaring in her head. That night her left upper lip felt slightly numb and the next day the entire left side of her face was similarly involved. She was unable to taste on that side of her tongue. The left side of her face appeared drawn. She was unable to coordinate movements of her left hand. These symptoms continued until shortly before admission when she began to notice a decrease in the roaring in her ear, improvement in hearing, decrease in dizziness, and return of sensation to the left side of her face.

Examination on admission showed nystagmus on lateral deviation, diminished hearing on the left, decrease in sensation around the mouth on the left side, and slight ataxia and past-pointing of the left hand. Caloric reactions showed left canal paresis. Audiograms revealed a 20 to 30 decibel loss on the left side. X-ray studies of the skull were normal. Lumbar puncture showed an initial pressure of 30 mm. of spinal fluid which was clear but contained 360 red blood cells per cubic mm., mostly crenated, with 43 lymphocytes. The total protein was 49 mg. per 100 cc.; the gold curve was 2222210000. The Wassermann reaction was negative.

Operation was postponed because of an upper respiratory infection and in that interval her symptoms further regressed. Finally on November 6, 1946, the left cerebellopontine angle was explored. The auditory nerve appeared normal. Between the auditory and trigeminal nerves, lying adjacent to the pons and pur-

suing a course from the neighborhood of the proximal origin of the auditory artery toward the trigeminal entry zone, was an artery which was obviously abnormal. In the mid-portion of its longitudinal course at this level there was a bulbous dilatation, the medial edge of which was ragged and had evidently been attached to the pons. It was from this point of attachment that the bleeding had occurred. The dilated area was approximately 3 mm. long and 2 mm. in diameter. The artery was clipped proximal and distal to the aneurysm. In a follow-up visit on December 19, 1946, the patient had no complaints. On examination, no evidence of neurological deficit was found.

Comment: At the onset this patient presented evidence of a syndrome resembling Ménière's disease; ultimately the paraphenomena localized a lesion in the left cerebellopontine angle. The presence of crenated blood in the spinal fluid made one suspect a leaking aneurysm in that location. At operation this was verified. Aneurysms are only one type of vascular disorder seen in the cerebellopontine angle. We have observed the sudden onset of vertigo associated with entwinement of the eighth nerve around an aberrant vessel. Vertigo has also been described particularly by Holmes[2] and by Dandy[3] as occurring with impingement on the eighth nerve by arteriosclerotic vessels.

CASE 4.—*Multiple Sclerosis.* A 39-year-old female was first seen on December 3, 1946. At that time her sole complaint was vertigo of two weeks' duration. While away on a business trip, and two days before menstruation, she awakened at 1:00 a. m. with extreme vertigo of such degree that she felt compelled to hold to the sides of the mattress for support. After about 10 days of this extreme condition, slight improvement was noted. There was gradual, although slight, improvement in her vertigo when first seen two weeks after the onset. Slight scanning of speech was noted. When this was discussed with the patient, she felt that it had come on simultaneously with her dizziness but was improving. Subjectively she continued to feel drunk. There was no rotation of the environment but when she attempted to read fine print, there was a definite blurring. Her past history revealed that two years prior to the onset of her present illness there was dimness of vision with apparent central scotoma of the left eye for a period of ten days.

Neurological examination revealed spontaneous nystagmus to the right, primary optic atrophy on the left, right-sided pyramidal tract signs characterized by increased deep reflexes and suggestive toe signs, and absence of abdominal reflexes bilaterally. Caloric reactions with water at 68° F. produced a response bilaterally in about 20 seconds. The right drum was slightly dull and retracted. The Rinne test was negative on that side. The patient has been observed for more than a year. She has in that time had several exacerbations of vertigo and more recently a low-grade optic neuritis on the right. At the present time the latter symptom has cleared entirely. This patient is on Dicumarol therapy.

Comment: This case, only one in a large group with multiple sclerosis, is presented because the patient's primary complaint when first seen was vertigo with nystagmus of a severe degree. Many other patients with this illness either have vertigo as an initial complaint or note its occurrence during the course of the illness. It is

our belief that when this symptom presents itself in a young adult with no hearing loss, the diagnosis of multiple sclerosis should be considered. Neurological examination of the patient might well reveal other evidence of the illness. With present methods of treatment, there is hope of preventing progression of multiple sclerosis. Early diagnosis is, therefore, essential.

CASE 5.—*Vertiginous Epilepsy.* A 51-year-old, married male was first seen on March 26, 1946. His history revealed that on October 20, 1945, while in a grocery store, he suddenly felt dizzy and fell to the left. The room appeared to be rotating clockwise. The attack lasted five minutes leaving the patient weak. During the next six months the attacks recurred with increasing frequency until when seen a year ago he was having these spells twice weekly. Following each attack the patient would become drowsy, lie down, and sleep for an hour or two. This suggested a possible association with epilepsy, so on April 16, 1946, an electroencephalogram was done. This revealed a moderately slow record, showing a significant amount of pace irregularity, but no focal signs, amplitude asymmetry or true paroxysmal activity. Occasional sequences of slow waves occurred on a pace irregularity background which were suggestively paroxysmal in appearance.

Neurological examination was essentially negative. When the caloric tests were done, the patient observed that the vertigo produced was an exact reproduction of his spontaneous attacks. No defect in hearing was found. The carotid sinus reactions were normal to compression. There was no histamine sensitivity.

Because this case suggested vertiginous epilepsy, the patient was given Dilantin Sodium, 0.09 gm. three times a day. In the following month he had three more attacks. His medication was changed on May 23, 1946, to Tridione, another anticonvulsant medication, 0.3 gm. four times a day. On this medication attacks were completely abolished.

Comment: It is fairly common to observe a feeling of rotation on the part of the patient at the onset of a convulsive seizure. Such rotation does not resemble vertigo in that it is of short duration. Diagnosis is simplified by the usual manifestations of a grand mal convulsion which follows immediately.

In the case presented here a vertiginous attack is the complete clinical manifestation of a convulsion. This type of disorder is unusual. The significant features are the paroxysmal vertigo with no symptoms or signs referable to auditory disturbance, the abnormal electroencephalogram, and the period of sleepiness following the attack. The latter is most important. The complete disappearance of attacks on anticonvulsant medication is significant.

CASE 6.—*Migraine with Vertigo.* A 22-year-old, single female was admitted to Barnes Hospital on January 17, 1947, complaining of vertigo, staggering gait, blurred vision, photophobia, and headache of several days' duration. Periodic sick headaches began in childhood. There was a family history of migraine on the maternal side. The headaches were confined to the right side of her head, above the eye and ear, and in the region of the occipital artery. They usually

lasted two to three days but could be aborted with ergotamine tartrate. With many headaches there was slight dizziness and blurring of vision. With recent attacks the latter symptoms had been much more severe. There was definite rotation of the environment with difficulty in standing in the last attack. It is noteworthy that for a period of over a year this patient worked as a nurse in an aviation evacuation unit evacuating wounded marines from Pacific Islands under fire. During that period she had fewer headaches than ever before. Since her return to civilian life emotional conflicts which had escaped resolution have come to the fore. With the increase in stress her attacks have become much more severe.

Neurological examination revealed a marked unsteadiness of gait with ataxia on finger-to-nose test bilaterally. Disturbances in coordination were noted in the left hand and the left lower extremity. She was unable to maintain her station in the Romberg position. The abdominal reflexes were absent. The deep reflexes on the left side were diminished. There were no pathological toe signs. There was a moderate nystagmus on lateral gaze to both right and left, but no other abnormal cranial nerve findings. Laboratory data, including cerebrospinal fluid tests were negative.

Comment: There is both a familial occurrence of migraine in this case and a history going back to early childhood. The attacks are characteristically unilateral, are relieved by ergotamine tartrate when given early, and usually terminate following vomiting. With many attacks a definite vertigo accompanied the latter part of the seizure. In the episode for which she was admitted to the hospital, the vertigo was of such severe degree that she was unable to walk. The temporal relationship between the two conditions leads to the conclusion that vertigo can be an accompaniment of the migraine attack. Several features in the case, that is, the absence of abdominal reflexes, ataxia in the finger-to-nose test, and nystagmus, suggested an acute onset of multiple sclerosis. There is some evidence to believe that the latter condition belongs in the category of paroxysmal disorders exemplified by this case.

Case 7.—*Migraine, Hypothyroidism, Vertigo and Personality Disorder.* A 49-year-old, married female entered Barnes Hospital on February 13, 1947, with a primary complaint of vertigo existing since November 1946. Until 12 years before admission the patient had had mild periodic headaches which began in adolescence. There had been progressive enlargement of her thyroid gland. Fourteen years ago this was removed. Thirteen years ago a hysterectomy was done and one ovary also was removed. She had no menopausal symptoms. One year later her headaches became more frequent and severe. At that time she learned that her husband was unfaithful; for years he had denied this. The patient felt hostile toward him but was unable to discuss the matter openly and to express her feelings. During this period there were no sexual relations between them. She carried on in this fashion with severe periodic headaches until four years ago when she sued for divorce after finding him visiting another woman. Nevertheless a reconciliation was brought about which her daughter felt was sincere on the part of the husband. In spite of this the patient remained unforgiving and rigid. Sexual relations were resumed but continued to be unsatisfactory. Insomnia developed associated with her anxiety and continuing distrust of her husband.

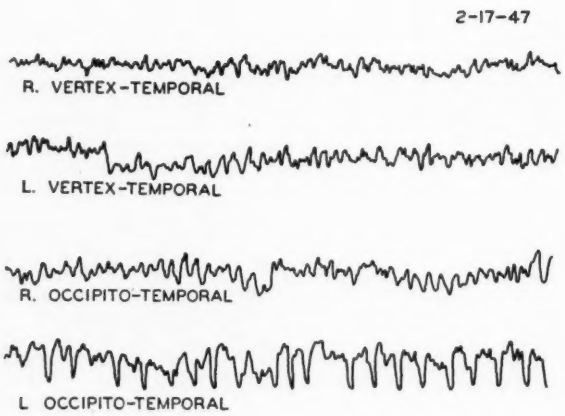

Fig. 5, Case 7.—Electroencephalogram of a patient with migraine, vertigo and hypothryoidism. There is a paroxysmal focus of flat-topped waves in the left occipitotemporal region.

Headaches became more and more severe. A nocturnal allergic rhinitis recurred more frequently.

Finally, two years ago, she had her first attack of vertigo. As she turned over in bed she stated that things started rotating about her from right to left. By morning this had disappeared. Three weeks later she had another attack in which she felt pulled to the left and objects again rotated about her. This lasted 20 minutes. In January 1946 another severe attack was followed by several minor episodes. In February 1946 while in New York, away from her husband, a severe attack occurred which kept her in bed. In the summer and fall of 1946 there were two or three more attacks lasting about 15 minutes each. In November 1946, when starting on a trip to Chicago for business purposes, she had a severe attack lasting all morning. Two days later, while away from home, there was a recurrence with vomiting. This was of severe degree and after that time there was persistence of dizziness in varying degree with mild bilateral tinnitus, a tendency to deviate to the left in walking and occasional vomiting. Some attacks of vertigo were accompanied by her typical migraine headaches. She was placed on a salt-free diet with no benefit. Three weeks prior to admission she was empirically placed upon thyroid, ¼ gr. three times a day, with moderate improvement.

Examination after admission to the hospital was negative except for the otologic and psychiatric findings. Her caloric tests with water at 68° F. gave a reaction in the left ear in 42 seconds, past-pointing 10 inches to the left, severe falling to the left, and nystagmus to the right which remained active for five minutes. In the right ear the nystagmus occurred in 35 seconds. There was nausea and explosive vomiting. She past-pointed 12 inches to the right, fell to the right, and nystagmus of a severe degree to the left was noted. Her hearing was good; the audiograms were normal. The Weber test was referred to the right. The Rinne test was positive bilaterally. The eustachian tubes were easily inflated.

Allergy skin tests showed general mild reaction to many substances. Her skin was dry and subjectively there was a feeling of coldness in the extremities.

Her blood pressure was 150/90. Electrocardiograms showed left axis deviation. X-ray films of the skull were negative except for underdevelopment of the mastoids bilaterally. In spite of the fact that the patient had taken thyroid in small doses for three weeks, her basal metabolic rate was -25; blood cholesterol was 255 mgs. Lumbar puncture showed an initial pressure of 125 mm. There were 2 lymphocytes, the protein was 35 mg. per 100 cc., the Wassermann reaction was negative, and the colloidal gold curve was 0000000000. Glucose tolerance test: fasting sugar 78 mg. per 100 cc.; one hour 128 mg. per 100 cc.; 2 hours 66 mg. per 100 cc.; three hours 52 mg. per 100 cc. The urine test was negative. The blood Kahn test was negative.

An electroencephalogram performed on February 17, 1947, showed the existence of a paroxysmal focus in the left occipitotemporal region. The paroxysmal waves were of moderate amplitude, frequency 3.5 to 4 per second, flat-topped, with intervening positive spikes (Fig. 5).

She was placed on thyroid gr. 2 daily, and numerous discussions of her problems were held. The latter revealed that her mother and father were divorced when she was a small child. Her father left her to the care of the mother who began to have affairs with numerous men. The child was sent to live with her grandmother, but was well aware of her mother's immorality and rejection of her. As a reaction to this the patient developed a very rigid, unforgiving personality with stunting of emotional development. Sexually she became frigid. These factors were the basis for her unhappy home situation and probably the cause for the extramarital activities of her husband. With treatment the patient improved.

Comment: Three points of interest are illustrated. First, the case demonstrates the occurrence of vertigo alone or in combination with headache of a migrainous character. Second, it demonstrates the role of thyroid activity not only in headaches but in vertigo. When the patient's metabolic rate is low and the cholesterol elevated, there is an increased incidence of paroxysmal manifestations. This can be corrected to a large extent by thyroid replacement therapy. The most important point to be learned from this case is the effect of emotion on this type of disorder. The suppressed hostility toward her husband in a situation which represented a re-enactment of the situation so often confronting her in childhood certainly affected the frequency and severity of headaches. Her suspicious, rigid, unforgiving personality aggravated the situation as well as did the fatigue resulting from the insomnia. One cannot expect to help patients of this sort without intensive psychotherapy.

CASE 8.—*Vertigo Followed by Anxiety Hysteria.* A 44-year-old, married male entered Barnes Hospital on February 11, 1947, for the third time. He was admitted first on April 6, 1937, at which time he gave a history of a tendency to fall to the right side and deviation to the right in walking of two years' duration. Nine months previously he had begun to have continuous tinnitus in the left ear with intermittent attacks of vertigo sufficient to make him fall to the ground. With these he sometimes lost consciousness. Because of these symptoms he was unable to work. At that time audiograms and caloric reactions were

essentially normal. He was placed on a salt-free diet and ammonium chloride was given by mouth. His complaint of dizziness remained fairly constant although it became milder in degree.

In May 1946 his attacks became more severe and frequent, forcing the patient to quit work and remain at home. There he was able to walk about without staggering. He could help his wife with the housework and wash and dry dishes. At no time did he have any difficulty, but if he attempted to venture out of the house his feeling of dizziness would appear. It should be noted that what he described at this time as dizziness was more a feeling of uncertainty and of impending disaster rather than a sense of rotation. If he went to the factory, he was unable to work because he staggered a great deal. He volunteered the information that he could drive the car with his wife in the seat next to him but could not drive alone. When questioned as to what would happen if his wife should leave him alone while they were away from the house he stated, "You would probably find me sprawled in the street."

All laboratory tests on this admission were normal. On strong suggestive therapy, small doses of barbiturates and benzedrine this patient has been able to return to work. A communication from him dated March 21, 1947, states: "I left the hospital the 18th day of February. I returned to work on the 24th of February. I have worked six days each week having only Sunday off. I do not have any trouble getting along with my work but do notice a stagger to the left when walking outside at times, but never notice the stagger inside the building."

Comment: This patient, with a history of constant vertigo, probably of a Ménière's type, for 10 years, was found finally to have developed a neurosis. In the past few years we have observed four patients whose symptomatology began as a true vertigo, usually sudden in onset. Gradually there evolved in each the pattern of anxiety hysteria known as "street neurosis." This is an extremely crippling disorder in which patients are not able to venture from the house because of fear of experiencing an extreme degree of anxiety.

DISCUSSION

Selected cases reported herein (Cases 5, 6, and 7)illustrate that there is an interchangeability between three of the known paroxysmally occurring manifestations, namely vertigo, headache, and convulsions. In the same patient two or all of these disorders may occur in sequence or simultaneously. Vertigo and headache are commonly associated; it is rare, however, for vertigo to be associated in the same individual with manifestations of a convulsive disorder. The family history in all cases of vertigo should be investigated for one of the other manifestations in order to define more accurately the nature of the disease process. We frequently observe a family history of migraine in epileptic conditions; the same is true in those cases of vertigo which fall into the category of migraine equivalents.

Vertigo may manifest itself in a paroxysmal fashion in one of several ways. The hydropic state of the endolymphatic sac may

occur paroxysmally. Then there is usually accompanying tinnitus and some diminution of hearing. This is the more common basis of paroxysmal vertigo and it is more closely allied to migraine than to epilepsy. Also, with no evidence of a hydropic state in the endolymphatic sac, vertigo occurs as a common accompaniment of the severe migraine attack (Case 6). More rarely, vertigo represents an atypical epilepsy and is independent from migraine (Case 5). The electro-encephalogram is frequently the diagnostic link between the paroxysmal disorders; it is of outstanding value when the interval record between paroxysmal attacks shows evidence of definite brain wave abnormality (Case 7), or when specific abnormal patterns appear which relate the background condition to either epilepsy or migraine.

Several factors influence the frequency and the severity of all paroxysmal manifestations. These are: emotion, fatigue, and coincident endocrine conditions. In emotional disturbances it is our experience that the hostility and anger which a patient is unable to express is frequently the important factor governing the acute upheavals in symptomatology (Cases 6 and 7). Frequently this hostility is just beneath the surface and easily ventilated by superficial psychotherapy with gratifying results. Unrelieved anxiety may act in the same way. Fatigue may be the precipitating factor behind a given attack, many patients tracing the onset of one or another paroxysmal episode to an unusual amount of effort.

The endocrine factors responsible for governing paroxysmal vestibular activity are principally gonadal and thyroid. It has long been recognized that the immediate premenstrual period is a time of increased irritability of the nervous system. At that time estrogen and progesterone levels are low. Hypothyroidism has been shown to alter the convulsive threshold.[4] This mechanism apparently acts through the migration of cholesterol away from tissue membranes and into the blood stream.[5] Aird[6] also injected cholesterol into animals and showed increase in the threshold for the action of convulsant drugs. There has been recent experimental work on ketosteroids to show their relationship to vasomotor phenomena and their increased excretion during attacks of migraine.[7] An increased outpouring of 17-ketosteroids has been shown to be associated with emotional stress.[8]

In the treatment of vertigo as well as that of the other paroxysmal disorders, these three factors, emotion, fatigue, and endocrine disorders, should be taken into consideration. One cannot success-

fully treat any paroxysmal disorder without psychotherapy. The patient should have ample opportunity to ventilate his feelings. Any endocrine dysfunction should be corrected. The basal metabolism should be brought up to within normal limits by thyroid therapy if it is subnormal (Case 7). Proper education in how to live with avoidance of excessive stress and fatigue should be incorporated into the regime of living. Occasionally, when the vertigo is closely allied to epilepsy, one of the anticonvulsant drugs may be of inestimable value in controlling the attacks (Case 5).

Other individuals with vertiginous symptoms may require more intensive psychotherapy than is suggested above. In Case 8 the patient had episodes of vertigo gradually evolving into a neurosis. In other words, the vertigo was the nucleus and set the pattern for the neurosis. Psychotherapy in such cases may be directed toward revealing those factors in the life history which influenced the development of the abnormal personality pattern. This is frequently a difficult and time-consuming process requiring a skilled therapist.

Several lines of investigation point to specific areas of the cerebral cortex as being concerned with vertiginous experiences. Foerster[9] stimulated the superior lip of the interparietal sulcus electrically in man and elicited violent vertigo with the sensation of turning toward the opposite side. Penfield and Erickson,[10] by electrical stimulation in approximately the same locality, produced the sensation of falling off the table without accompanying movement. Others have reported an incidence of vertigo as a neighborhood symptom in tumors involving the temporal lobe and opercular region of the frontal lobe.[11] Experimental evidence[12] relates the temporal areas of animals to the vestibular nerve; after strychninization of this area rotation of the animal produced convulsive attacks. Electroencephalographic changes have also been reported in the temporal areas of experimental animals following rotation.[13] Focal pathology in the above-mentioned cortical areas should be kept in mind in considering tumors as a possible etiological factor in the causation of vertigo.

Within the brain stem the most common causes of vertigo are the acute vascular accidents, multiple sclerosis, disseminated encephalomyelitis and encephalitis. The history and paraphenomena associated with each of these point to the diagnosis. In evaluating vascular accidents in the brain stem it is necessary to be familiar with the pattern of vascular distribution in relation to different tracts and nuclei in order to make a diagnosis of occlusion. The vestibular

nuclei, for example, are supplied by the long circumferential branches of the vertebral-basilar system. One such branch is called the posterior inferior cerebellar artery. In addition to the vestibular nuclei, the spinal tract of the trigeminal nerve, the spinothalamic tract, the nucleus ambiguus, pupillodilator path and a part of the inferior cerebellar peduncle fall within its distribution to the brain stem. Occluding lesions of this vessel may produce vertigo with ipsilateral lateropulsion, hypalgesia and hypesthesia of the ipsilateral side of the face, paresis of the ipsilateral palate and vocal cord, ipsilateral Horner's syndrome and contralateral analgesia below the neck.

Multiple sclerosis gives rise to vertigo of sudden onset but will usually occur at a younger age than is common for vascular occlusions. In addition the lesions of multiple sclerosis seldom give evidence of neurological deficit that could be related to occlusion of a single vessel. The existence of other evidence of multiple sclerosis such as absent abdominal reflexes, spastic-ataxic gait, intention tremor or central scotoma serves to confirm the diagnosis.

Disseminated encephalomyelitis is frequently difficult to distinguish from multiple sclerosis but is generally considered to be an acute widespread inflammatory reaction involving the brain stem by perivascular demyelination. The clinical manifestations frequently give evidence of involvement of the vestibular system. In encephalitis the type may be undetermined but the co-existence of eye muscle paralysis suggests the von Economo type.

Brain stem reflexes may arise from excessive stimulation of the vestibular nuclei. Nausea and vomiting are the most frequent autonomic manifestations observed. Strong vagal reactions with pallor, coldness and sweating are frequently seen. In some individuals the vagal response may be so strong as to produce syncope (Case 2). Ipsilateral hyporeflexia may result from vestibular stimulation.

In our experience vertigo has not been a particularly useful symptom in contributing to the diagnosis of cerebellar lesions. Holmes[2] apparently shared a similar experience.

In the cerebellopontine angle acoustic neurinomas cause vertigo infrequently; on the other hand, tumors of different types may well give rise to vertigo either as an initial symptom, or the vertigo may appear later in the development of symptoms (Case 2). Infections involving the angle may be pyogenic and result from the spread of an old ear infection with subsequent walling off of the infectious

process. Granulomas, such as those of syphilis and torula, have some degree of predilection for this area and usually produce vertigo among other manifestations. The pathologic condition in the granulomatous lesions may be bilateral. The spinal fluid is the significant feature for differential diagnosis between them. In syphilis there will be an increase in cell count and protein, the Wassermann is usually positive, although it may be weakly so. The colloidal gold curve more often shows some degree of midzone rise. In torula meningitis the spinal fluid sugar and chloride decrease; budding yeast forms can be seen in the hanging drop, and the organism grown on Sabouraud's medium.

SUMMARY

An attempt has been made to show that a central disturbance that has vertigo as a symptom is diagnosed by evaluation of the paraphenomena associated with it, rather than by the characteristics of the vertigo itself. Eight cases are presented illustrating different problems in diagnosis, and the type of treatment is indicated for each.

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LII

SUMMATION

SYMPOSIUM ON VERTIGO

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To prepare a summation of the material so completely and brilliantly presented by the essayists is to accept a very responsible task. An initial inquiry into their discussions reveals an extreme breadth of thought and understanding of the subject and baffles any attempt to bring it together in a readable and coherent summary. Obviously, I cannot refer specifically to all the major issues that have been posed in this panel. I can only hope to select a few of the "high spots" as a record in abstract of those observations and experiences with which I can claim some personal contact.

The importance of contemporaneous studies is something of which we become aware when listening to a discussion of this comprehensive topic. Our knowledge of vertigo has not been obtained and developed as something purely otolaryngological and distinct and separate from the other sciences. This point has already been illustrated by the necessity of bringing together some sincere and honest men from the ranks of other fields of medicine to direct opinions to the real issues and give us enlightenment on this subject.

I should like to take your time to discuss a few clinical manifestations of vertigo, those which come to our attention almost daily in the routine practice of our specialty. For practical purposes—everyday usage, as it were—I shall select those references from the previous discussions that might well be emphasized before an audience of clinical otologists.

It is appalling the number of patients who seek the services of an otologist because they are annoyed by the symptom of dizziness. It is equally amazing how infrequently one is able to find an explanation for this condition. These patients often appear to be in perfect health; perhaps they complain only vaguely of headache, weakness and nervousness but fail to show any organic reason for their symptoms. They do not experience violent vertiginous attacks

such as those observed in true Ménière's disease; rather do they complain of an unsteady or giddy feeling which is more or less constant, but aggravated by changes of posture.

As described by Dr. Lewy this symptomatology no doubt is the result of a toxic factor in some patients, although we have little pathological evidence of its existence or the site of its influence. Examples of this type of vertigo in an aggravated form are occasionally seen in patients who have been profoundly septic, as in individuals convalescing from pneumonia, typhoid fever, or erysipelas. We can hardly question the occurrence of a toxic form of vertigo as an entity and it is highly probable that some patients who complain of an unsteady feeling or mild fleeting dizzy sensations without impaired auditory function are experiencing the effects of toxic absorption from some obscure source seldom revealed by a most thorough physical examination.

There is no pathological evidence, however, to justify the diagnosis of toxic labyrinthitis in these patients.

A number of years ago we failed to find pathological changes in the otic labyrinth in patients who had died from typhoid fever. The morbid changes likewise were not found in microscopical sections from the acoustic nerves. It is doubtful if the clinical manifestations which we have attributed to a toxic labyrinthitis can be definitely ascribed to any isolated part of the auditory tract; rather it is more accurately assumed perhaps that any or all of its constituents may participate in changes responsible for this symptomatology.

I point with gratitude to the clear and comprehensive description given by Dr. Levy and Dr. O'Leary of another group of patients in this category that comprise a goodly portion of those who daily seek professional advice. Their chief symptom is dizziness, but we also find in them indications of a nervous instability which clearly reveals a psychological state in the nature of a depression. This affliction is well known to all of us, yet frequently is most difficult to differentiate from an organic illness. The patient is tired, unable to work, complains of insomnia, is depressed and has lost interest in living. These depressions are often self-limiting but in many instances they call for the advice and care of the trained psychiatrist. The dizziness is often persistent and worthy of sounder therapy than that which is frequently offered by the administration of one drug after another or gained by frequent visits to different physicians, eustachian tube inflations and innumerable operations upon the nose and throat. It becomes a matter of much importance to recognize

the fundamental etiological factors in this disorder and administer effective treatment. The resources and skill of the psychiatrist are required and it is to him that we must look for the proper therapeutic management of this group of patients.

The more I see patients in the daily practice of medicine who complain of dizziness, the more I am impressed with the emphasis given by Dr. Levy and Dr. O'Leary to the frequent presence of functional manifestations. Psychical disturbances assume a major role in many of them. I venture to state that there is often a large psychical element in patients with the so-called typical Ménière's disease. It is probably the effect, not the cause of this syndrome; nevertheless, it is often conspicuously present and its influence as a contributing factor in the patient's illness is most difficult to evaluate.

Come now to a third, but much smaller group of patients, namely those with hypertensive disease who complain of dizziness. They also demonstrate some degree of auditory impairment although they seldom appear to be concerned with this defect. They are much more troubled with a persistent giddy sensation and occasionally a tinnitis. From a rather large series of patients with hypertension we observed that the majority of them had impaired hearing of a perception type which they had not noticed and one third of them complained of tinnitus and some degree of dizziness. In so far as we could determine, there appeared to be no correlation between the severity of vertigo and the degree of hypertension. Individuals with excessively high blood pressures often complained of only slight sensations of dizziness, while patients with hypertension of a mild character frequently evidenced a greater disturbance of equilibrium. By the same token there appeared to be no parallelism between the duration of the hypertension and the degree of vertigo.

An opportunity for further study of deafness and vertigo in hypertensive disease was afforded in 37 patients of our series who submitted to splanchnicectomy as a surgical means of reducing the blood pressure. Audiograms were made three months after the operation and compared with the preoperative charts. It was interesting to note that in most patients there was a striking decrease in the hypertension after the operation and that this was associated with some improvement in hearing and a disappearance of the vertigo. The gain in auditory acuity affected the high tones but was not observed in the lower tone range. Of striking significance, however, was the disappearance of the vertigo; this is obvious evidence that hypertensive disease acts somewhere upon the equilibratory mechanism

to produce vertigo and yields admirably to surgical measures that are successful in accomplishing an appreciable reduction in the blood pressure.

The best way to diagnose this type of vertigo is to keep the possibility of hypertensive disease constantly in mind. The symptoms, headache, dizziness, tinnitus and nervous irritability, aggravated by fatigue and emotional strain should furnish suspicious evidence of this disorder and the diagnosis will ultimately be revealed by a series of sphygmodynamometer determinations. It must be emphasized, however, that the physician cannot base a positive diagnosis of hypertension upon one sphygmodynamometric reading. The disease may evidence a fluctuating character in its early stages and one must take several determinations at varying intervals before concluding that the patient's blood pressure is within the normal range.

On several occasions in a lifetime of practice the otologist will be consulted by a patient who suddenly, without provocation, suffered an explosive attack of vertigo with a "roaring" or "pounding" noise in one ear, nausea and vomiting and a profound unilateral deafness. The symptoms are apoplectic in onset, often exceedingly violent and totally incapacitating for a period of ten days to two weeks. The severity of the symptoms made an unforgettable impression upon me on one occasion when I was called to rescue from the hands of the police a member of the clergy who was a victim of this condition. Although characteristically attired, and in spite of vigorous protestation, he was nevertheless regarded as completely out of case and about to be booked in the police courts for intoxication.

Usually within two or three days the nausea and vomiting cease; ten days later the dizziness disappears and although the latter may persist in a mild form for months, or even years, a high degree of permanent hearing loss is the rule.

The pathological change responsible for this type of vertigo is not definitely understood although it is assumed to be the result of a vascular accident within the otic labyrinth. This would seem to be quite consistent with the findings and course of the disease since the precipitous and violent character of the symptoms are definitely those due to stimulation of the end-organ and the permanent effects upon the auditory function those which might be expected from circulatory interruption to nerve centers.

Under some circumstances the otologists will be consulted by the patient with an acoustic neuroma. Vertigo is not usually a prom-

inent symptom of this lesion although is is occasionally observed in a mild form. The early indication of a tumor of the auditory nerve is impaired hearing and it is frequently accompanied by other neurological manifestations when the patient first consults the doctor. Thus the services of the neurologist or neurological surgeon are initially sought. It is amazing how often the patient with impaired hearing delays consultation with the otologist for months or even years before he becomes concerned about his disability. A visit from the patient with a profound deafness of ten or more years' duration and expectations from him that immediate curative measures can be instituted is not an uncommon experience. The patient with an acoustic neuroma is no exception to this rule. He is little concerned with his hearing defect until it is accompanied by symptoms which call for a neurological examination, pain over the distribution of the inferior division of the fifth nerve, weakness of the muscles of mastication, facial paralysis, extraocular palsies, visual disturbances and signs of intracranial pressure.

Dr. Lindsay has called our attention to an important group of patients with vertigo due to inflammatory reactions within the otic labyrinth who are frequently seen in an otological practice. Perhaps they have had a chronic discharging ear for many years which caused them no particular annoyance until recently they experienced some severe paroxysms of dizziness. A spontaneous nystagmus may be present. Occasionally the fistula test gives a clue to the nature of the lesion. At operation one finds a fistula usually perforating the external wall of the horizontal semicircular canal leading into the labyrinth. Rarely are formidable labyrinthine operations required for these patients. A well performed radical mastoidectomy and the administration of the chemotherapeutic agents and the antibiotics will usually suffice to bring about a satisfactory recovery.

And finally we come to a consideration of the vertigo observed in a typical Ménière's seizure. Dr. Lindsay's work in this field has been colossal. He has revealed the pathological changes responsible for this condition and created an epoch in otological progress that will not fail of listing recognition in the end. Let us not be too precise and academic about the designation Ménière's disease. Perhaps it is a term loosely applied and much abused; even a misnomer. Nevertheless it deals with an otological concept, one that among otologists does not need redefinition. It conveys to us a symptomatology not to be confused with that of any other disease; an attack of vertigo in a patient with unilateral deafness; occasionally impaired hearing on both sides but more profound in the ear to which the

symptoms are referred; nausea, vomiting and spontaneous nystagmus in the severe seizures; tinnitus, often unilateral, increased just before and during a paroxysm; occasionally a pathetically disabled patient suffering from a violent vertiginous attack or living in constant terror of an impending one.

I have frequently made the statement that the vertigo of Ménière's disease is unlike that produced by any form of intracranial disorder, brain tumor, abscess, multiple sclerosis, cerebral arteriosclerosis or vascular accidents. The typical symptom is a violent vertiginous seizure, paroxysmal and often disabling to the extreme of felling the patient in his tracks. The vertigo of intracranial disease is more of the character of a "swimming," giddy or unsteady sensation, present most of the time but aggravated by sudden movements of the body. The latter should not be mistaken for the vertigo of Ménière's disease. To me this differential point is exceedingly important. From my own convictions and the support of critical judgments of several neurological surgeons who are authorities in their field in this country, I emphasize the significance of the type of vertigo in Mèniére's disease as contrasted with that observed in the intracranial lesions to which we have alluded.

In Dr. McNally's customary brilliant and erudite manner he has given us a most complete discourse on the physiology of the vestibular mechanism in relation to vertigo. Among the many instructive concepts which he has offered one stands out as particularly appropriate in this discussion. He states, "In deciding upon the selection of a suitable vestibular test it is important to maintain a common sense outlook." And may I add that this also holds true for the interpretation of a vestibular test. Complicated tests involving anatomical pathways about which we are still much in the dark and the allocation of important diagnostic values to them are destined to lead the examiner astray in many of his diagnostic efforts.

CONCLUSIONS

1. Vertigo may be a symptom of some toxic disorder. Its occurrence in septic patients, after the administration of certain drugs and more recently of the antibiotic streptomycin, is considerable proof of this assumption.

2. Vertigo may be one of the manifestations of a nervous depression. The diagnosis is usually not difficult, although a satisfactory therapeutic response is not always obtainable. In this connection a word of caution is offered. The otologist must be careful not

to do or say something that will fix the patient's symptoms upon some specific cause. When one offers a precise explanation for the patient's ills and assures successful results from treatment or an operation, there is always the risk of a therapeutic failure and the permanent crystallization of the patient's symptoms. The sedative Dilantin has seemed to benefit some of these patients.

3. Vertigo is occasionally a symptom of hypertensive disease. The prospect of relieving the patient of this troublesome condition is favorable if surgical measures result in a substantial reduction in blood pressure.

4. Organic lesions of the central nervous system may produce vertigo but they are usually associated conspicuously with other clinical signs of intracranial disease. The classical example is the cerebellopontine angle tumor.

5. There are instances when vertigo is the result of an end organ lesion in the acoustic nerve. The vertigo derived from this source, as for example that of Ménière's disease, is so specifically characteristic as not to be confused with any other type. I speak succinctly and without fear of derision that if the patient suffers a violent paroxysmal vertiginous attack—one that fells him in his tracks—we are dealing in all probability with an end organ lesion, not an intracranial disease.

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LIII

PATHOLOGY OF THE LARYNX

A PHOTOGRAPHIC ANALYSIS

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The purpose of this paper is to present a photographic record of some of the more common types of laryngeal pathology. The gross appearance of the larynx is shown as it is seen with the laryngeal mirror or through the direct laryngoscope. Beginning the series of photographs are indirect views of the normal larynx, followed by the direct views of the landmarks visualized during the introduction of the laryngoscope. Subsequent photographs are those of individual cases. A photomicrograph of the biopsy specimen and in two cases photographs of the laryngectomy specimen are presented with the laryngoscopic picture to correlate the gross and microscopic pathology. A brief history, the histological diagnosis and the microscopic description are given for each case.

A description of the camera, the laryngoscope and the technique used to obtain the photographs is included because of the widespread interest of laryngologists in laryngeal photography. The photographic factors are included with each case report. The first number within the parentheses indicates the size of the General Electric flash lamp; the second number preceded by the letter *f* indicates the *f* stop, and the last number indicates the photographic laryngoscope used.

The camera* (Figs. 1 and 2) consists of an apparatus, held by a pistol-grip handle, to which various special flanged open-tube endoscopes or appropriate mirrors may be attached.[1, 2] The camera lens, diagonal mirror for the flash bulb, telescope, and focusing lamp, to-

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*The endoscopic camera used was constructed under the auspices of The Jacques Holinger Memorial Fund.

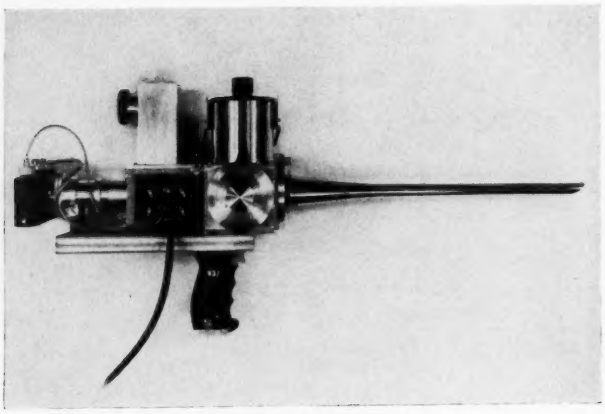

Fig. 1.—Right side of camera with laryngoscope attached.

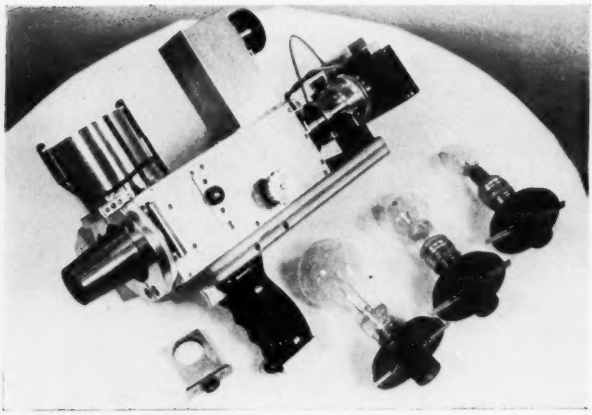

Fig. 2.—Left side of camera showing glass slide, focusing knob, and supplementary lens slot. Bakelite caps are shown with two adaptors for small bulbs.

gether with the releasing trigger, handle, and other associated parts, are built as a functional unit, and the camera film box with focal-plane shutter is permanently attached to it. The camera lens is a Zeiss 15 cm. (5⅞ inches) focal length *f* 6.3 Tessar in a barrel mount with iris diaphragm. It is mounted to permit rack-and-pinion focusing through a distance of 2 inches (50 mm.) total travel. The lens stop is adjusted by an external knob on the case, *f* stops of 9, 12, 18, 25 and 36 being provided. The focusing motion permits placing the object plane of sharp focus at any distance from 13 to 20 inches beyond the attaching clamp, as seen in the telescope.

A slot in the left side of the housing permits insertion of a supplementary achromatic lens of 11¼ inches (284 mm.) focal length ahead of the photographic lens. A dummy lens carrier is used to fill up the slot while photographing in the 13- to 20-inch range. The supplementary lens permits photography at 1 to 1 ratio (life size) at a distance of 5 inches (13 cm.) beyond the attaching clamp. This distance can be used for photography of the ear drum (Fig. 6), inside a nostril, or for photographing a small area in the mouth which is not over 1 inch (25 mm.) in diameter.

A 45-degree mirror is used behind the camera lens to direct all the light rays to the telescope system. The mirror drops out of the way by action of the trigger the instant before the picture is taken. This 45-degree mirror, as well as the porro prisms in the telescope-erecting system, consists of a front surface aluminum coating on the hypotenuse of a right prism. The optical reflection takes place on the aluminum coating and the rays do not enter the glass prism. This is done to avoid the aberration which would be produced if the rays traversed solid glass porro prisms. This aberration would be equivalent to that produced by a glass plate about 2 inches thick. Thus the full sharpness of the photographic image, just before the picture is taken, is seen in the telescope eyepiece. The full theoretical resolving power of the lens is obtained. Instead of a ground glass focusing screen in the camera, the reflex mirror reflects the image before photography to an eyepiece of a telescope type focuser which uses the camera lens as the telescope objective lens. No ground glass is used at the eyepiece focal plane. Experience has shown that the low contrast often seen in light and dark areas of the endoscopic field can be focused upon more accurately by using the aerial image instead of a ground glass image. The eyepiece shows detail as small as .001 in diameter.

An important part of the telescope system is the porro erecting system which produces an erect image of the field at the tip of the

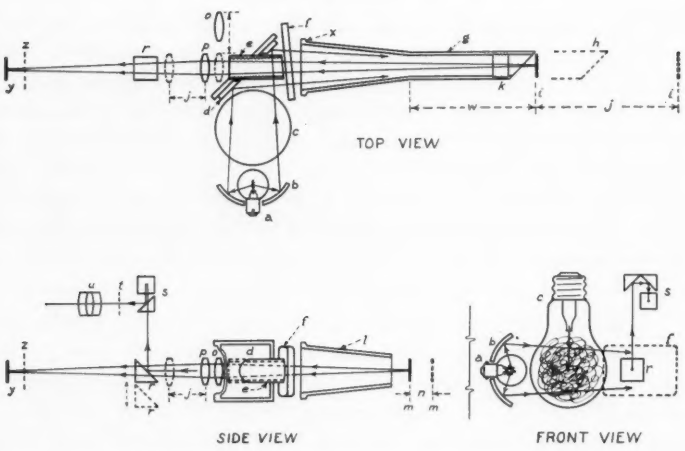

Fig. 3.—Optical drawing of camera (diagrammatic). *a*—focusing lamp, 6 volt, 32 c.p.; *b*—parabolic mirror; *c*—wire-filled flashbulb; *d*—45 degree plane mirror; *e*—tube, threaded on inside; *f*—optically flat glass slide; *g*—laryngoscope; *h*—tip of bronchoscope or esophagoscope; *i*—plane of sharp focus at object; *j*—range of focus adjustment of camera lens; *k*—threads to produce light ring around image on film; *l*—short tube to direct light at supplementary distance; *m*—object plane in sharp focus when supplementary lens "o" is used; *n*—range of focus adjustment with supplementary lens; *o*—Achromatic supplementary lens, 28.6 cm. focal length; *p*—camera lens, *f*6.3 tessar, 15 cm. (5 7/8 inch) focal length. Usable *f* stops from *f*.9 to *f*.36; *r*—Mirror to reflect light to focusing telescope. Moves out of way when picture is being taken; *s*—Porro erecting system; *t*—image-plane at eyepiece; *u*—eyepiece lens; *w*—free working length of laryngoscope; *x*—attaching flange; *y*—film in Leica camera box; *z*—focal plane shutter in camera box.

endoscope. It is important to have this image erect and correct from side to side, so that observation through the telescope focuser will be similar to looking through the open-tube endoscope itself. This eliminates the danger of trauma to the air and food passages which might result if the image were upside down or transposed from side to side, as it would be if the erecting system were not used.

The illumination for photography is obtained from a No. 50 or a No. 22 General Electric flash bulb, the light from which is projected onto the axis of the endoscope by a diagonal mirror with a hole and a light-shielding tube in its center. A 32 candle power, 6 volt automobile lamp, with parabolic mirror, projects light through the wire-type flash bulb and down the endoscope to provide light by which the endoscopic area can be seen during introduction of the endoscope into the patient. It provides sufficient illumination for selection of the area to be photographed and for critical focusing on the detail which is to be shown in the finished picture.

A heated glass slide is used between the diagonal mirror and the endoscope to shield the optical parts from condensed moisture

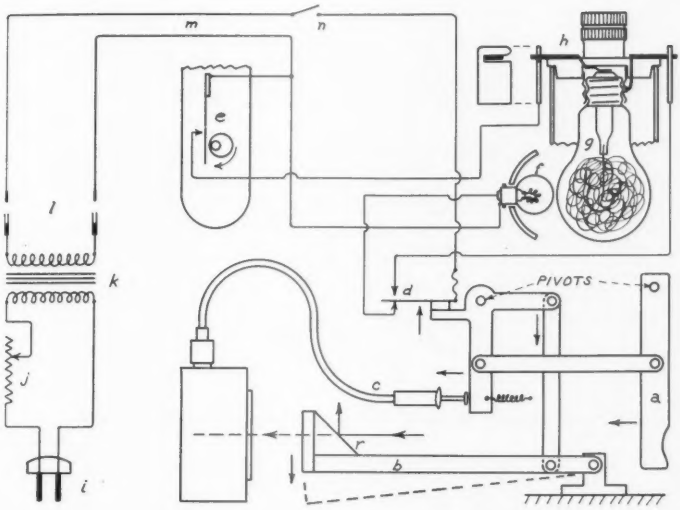

Fig. 4.—Electrical and control diagram of camera, *a*—trigger in handle of camera; *b*—lever carrying mirror "r"; *c*—cable-release to Leica camera box. Focal plane shutter set at "bulb"; *d*—contacts to control current to focusing lamp and flashbulb; *e*—contacts on top of camera box. Cam closes contacts just as shutter opens; *f*—focusing lamp, 6 volts, 32 c.p.; *g*—flashbulb (wire-filled type); *h*—removable bakelite socket cap, carrying flashbulb, with bayonet connections to the electrical circuit; *i*—connector plug for 115 volts a.c.; *j*—rheostat for adjusting focusing lamp voltage (7½ volts); *k*—40-watt transformer (10-volt secondary); *l*—connector; *m*—cord 10 feet long to camera; *n*—off-on switch; *r*—Mirror to divert light to telescope focuser.

and from soiling should the patient cough. A rectangular opening at one side of the front face of the glass slide permits free passage of air through the endoscope so the patient can breathe freely when the bronchoscope or direct laryngoscope is used.

The camera portion of the apparatus is essentially similar to a reflex camera with a focal-plane shutter. The camera film box is a standard Leica model, without range finder or lens. The automatic double-exposure prevention is a valuable feature. The focal-plane shutter of this camera simplifies some of the mechanical problems of design of the apparatus. A special electrical contact box is permanently mounted in the camera range finder clip, and engages a cam on the shutter-speed setting knob. The flash bulb is fired at the instant the camera shutter becomes fully open. The camera shutter is used on "bulb." On this setting the shutter opens when the release button is pressed by action of the trigger and closes when the button is released. Thus the open-shut flash method is used, utilizing all the light from the flash bulb (1/25 sec.).

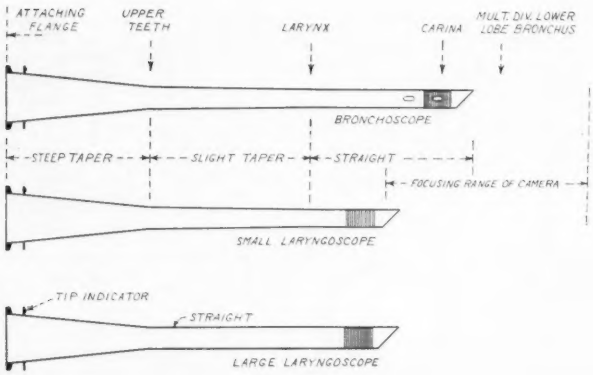

Fig. 5.—Sectional views of endoscopes (approximately to scale)

The trigger mechanism and associated parts operate automatically and are self-resetting. The only operations necessary to prepare for taking the next pictures are to replace the flash bulb, and to advance the film-winding knob to the next picture which in addition automatically resets the focal-plane shutter. The trigger lever in the handle is connected by levers to: the telescope reflex prism lever, the focusing lamp switch, the flash bulb switch, and to the cable release which opens the Leica camera shutter (Figs. 3 and 4). The focusing lamp produces a light value of about 40 foot candles reflected light (Weston) at the endoscope tip. The lamp is automatically switched off just as the trigger starts its travel. As the trigger is pulled further, the telescope reflex prism drops out of the light path from lens to film, and the current is connected to the flash-bulb circuit ready for the contact on the film box to fire the flash bulb just as the shutter opens. At the end of the trigger travel, the shutter-release button is pressed by means of a built-in cable release connected to the trigger lever. All of this trigger motion takes place within about 1/10 second. The current to the flash-bulb circuit is turned off except when the trigger is pulled. This is to prevent accidental flashing of the flash bulb if the bulb is replaced before the film is advanced for the next picture.

The full flash duration of the No. 50 bulb is utilized. The total flash duration is about 1/20 second, but 90 per cent of the light occurs during an interval of 1/25 second, and for all practical purposes the exposure time can be considered to be 1/25 second.

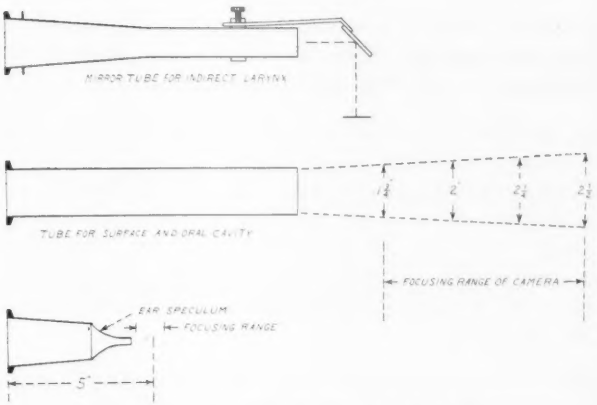

Fig. 6.—Sectional views of light-reflecting tubes used for mirror laryngoscopy, the oral cavity, and the ear drum.

A valuable feature is the method of flash-bulb loading. The flash bulb is used in an inverted position, the base being screwed into a bakelite cap. The bakelite cap has two insulated metal blades which engage two insulated metal plates or ears on the flash-bulb housing tube. Thus a bayonet type lock is used. Three duplicate bakelite caps are used, one in the camera, and two are kept loaded with flash bulbs ready for instant use. This allows reloading the flash bulb and advancing the film in about 3 seconds, ready for taking the next picture.

The endoscopes used are of special construction (Figs. 5 and 6). They are designed to take advantage of the largest possible diameter at the upper teeth and taper to the smaller diameter at the vocal cords. The endoscopes are polished and nickel-plated on the inside to preserve the high reflectivity of the surface. This inside surface near the tip is threaded for a short distance to provide a ring image on the film by which a circular mask can be located to mark off only the circular field wanted. The endoscopes are heated slightly just before use by a brief immersion in the sterilizer. Condensed moisture on the inner surface would reduce the reflection of the light from the flash bulb, the individual droplets of moisture acting as a rough surface. For the same reason, the glass slide for the camera is heated just before use by an alcohol lamp, and the slide is carefully cleaned after heating. The focusing lamp keeps the glass slide warm until the camera is actually used. A spare glass slide is constantly kept warm, ready for use, should the patient cough and spot the glass slide in the camera.

Technique. The endoscopes are introduced into the patient with the camera attached, since the focusing lamp and telescope give full visualization of the field at the endoscope tip.

The quick-acting attachment clamp for the endoscopes provides instantaneous release of the camera so that secretions can be removed by suction when necessary. In order to observe the endoscopic field when the camera is detached, an independent light carrier of the proper length is used, held inside the scope. No light-carrier canal or suction tube is permanently built into the endoscopes, since it would encroach on the circular field and spoil the appearance of the finished picture.

While the endoscope is being introduced, the plane of sharp focus is set just beyond the tip of the endoscope. Focusing for photography is done after the desired field is located. The depth of focus is adequate to show the local area in very sharp detail. Areas at some distance away are only slightly out of focus and most endoscopic areas cause no particular trouble when the finished film is viewed. Some judgment is necessary in choosing each area to be photographed, since pictures without easily recognizable landmarks have little value.

The *f* stops used (marked on the lens and not taking the lens extension into account) vary from *f* 12 to *f* 25 with *f* 18 about average for the larynx and tracheobronchial tree. The lens setting can be predicted according to past experience to better than one-half stop, so that the results are totally predictable with Kodachrome Type A film.

Motion of the field, especially in photography of the larynx, trachea and bronchi, requires careful judgment as to the exact instant for taking the picture to avoid excessive motion and blurring of the pictures. Vibration of the camera due to rapidly pulling the trigger release is not a serious factor, but some care must be used. Since the whole apparatus from tip of endoscope to film consists of a rigid structure, there is no problem of motion of the camera in relation to the endoscope tip.

Experience has shown that if much of the field is covered with blood, the pictures are not satisfactory because the red color of the blood is reflected into the other areas and distorts the true color values in the finished film (Fig. 34).

The alternating current from the well insulated low voltage transformer provides 5 amperes at 7½ volts. This circuit is used both for the focusing lamp and for the flash bulb circuit. No batteries are used.

The photographs of the larynx were originally 35 mm. Kodachrome Type A., color transparencies. In order to make black and white prints it was necessary to rephotograph the transparencies producing a black and white negative from which the enlargements were made. Extreme care and patience were necessary to maintain contrast and sharpness of focus in the final prints. It would have been quite simple to use black and white film, but the laryngeal pathology shows better in color and is more advantageous for visual presentation. The time required to take both Kodachrome and black and white photographs on each patient made this impractical.

SUMMARY

A photographic record of some of the more common types of laryngeal pathology is presented.

Black and white photographs of indirect and direct views of the normal larynx and the direct laryngoscopic views of the steps in the introduction of the laryngoscope are shown.

The laryngeal view with the photomicrograph of the biopsy specimen is presented to correlate the gross and microscopic pathology of 25 cases of hyperkeratosis, pachydermia laryngis, nonspecific and tuberculous granuloma, mucous retension cyst, polyp, papilloma and carcinoma. A description is given of a new still camera for endoscopic photography. The technique of its use and the photographic factors used in producing the various photographs are presented.

Photography of the larynx is no longer in the experimental stage. It can be seen that it is a valuable adjunct to the recording of clinical data, the presentation of cases and to visual teaching methods.

We wish to take this opportunity to acknowledge the work of Dr. Edwin F. Hirsch and Miss Inez Frischkorn of the Departments of Pathology and Photography, respectively, St. Luke's Hospital, Chicago, for their assistance in the preparation of the photomicrographs herein presented; and the work of Mr. J. D. Brubaker, for his preparation of the black and white prints of the larynx from the original kodachrome transparencies.

700 NORTH MICHIGAN AVENUE.

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2. Brubaker, J. D., and Holinger, P. H.: An Endoscopic Motion Picture Camera for Otolaryngology and Broncho-esophagology, J. Biol. Phot. Assn. 15:171 (June) 1947.

Fig. 7.—Indirect view of the normal larynx.

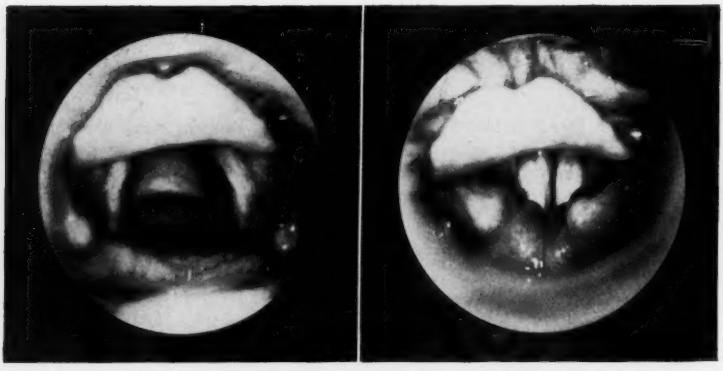


Fig. 8.—Indirect view of the normal larynx during inspiration.

Fig. 9.—Same larynx during phonation.

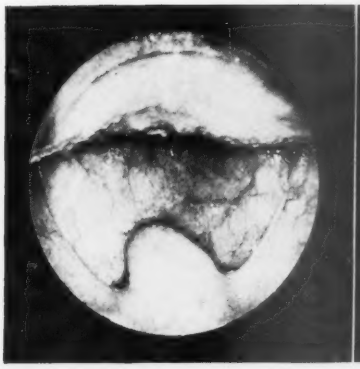


Fig. 10.—Base of tongue, posterior pharyngeal wall, uvula and posterior portion of the soft palate.

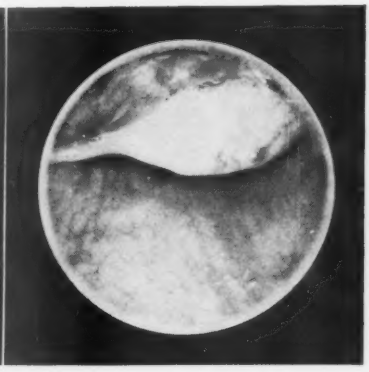


Fig. 11.—Base of tongue, tip of epiglottis and posterior pharyngeal wall.

Fig. 12.—Epiglottis lifted forward exposing posterior portion of the larynx.

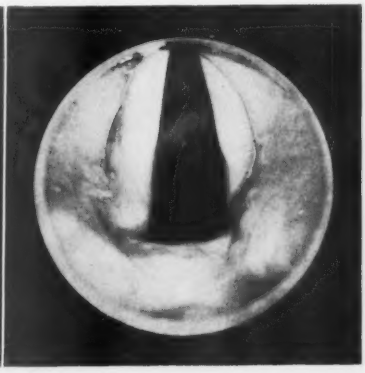
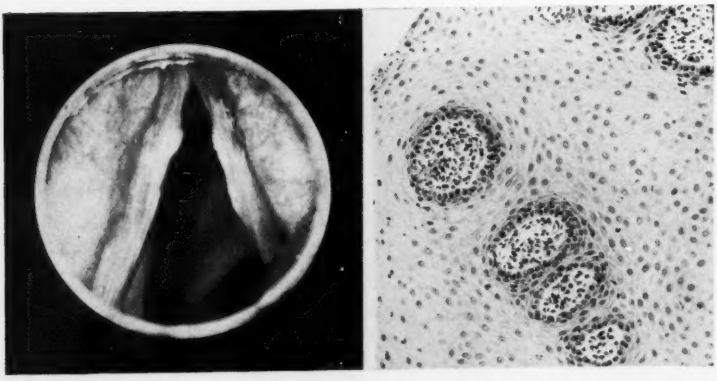
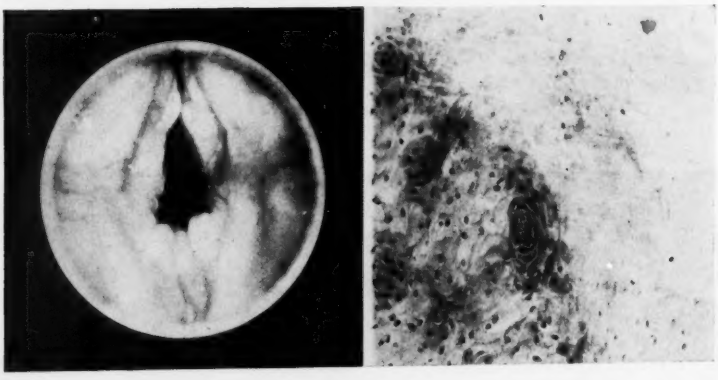


Fig. 13.—Larynx exposed.

Steps in the Introduction of the Direct Laryngoscope as Seen Through the Laryngoscope with the Patient in the Recumbent Position.





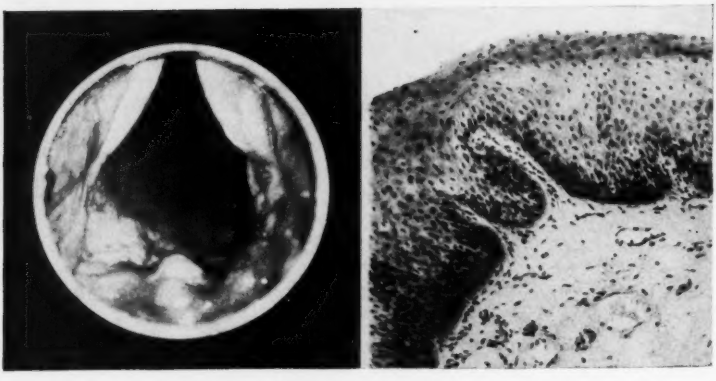


Fig. 14.—Hyperkeratosis of Larynx.

CASE 1.—White male, age 41. Film No. 63-10 (22-*f*18-3/4L).*

History: Hoarseness for 5½ months which started with a cold and sinusitis. Intermittent laryngitis but hoarseness persisted. Has had chronic sore throat.

Histological Diagnosis: Slight chronic inflammation and hyperkeratosis of larynx.

Microscopic Description: The squamous epithelium where cut sagitally has a surface hornified layer, and the fibrous stroma beneath it has exudates of lymphocytes and plasma cells. The epithelial tissues form a definite and sharp boundary and are not atypical. Another piece is cut horizontally to the surface and has considerably more epithelium of the squamous type in which there are scattered masses, generally small, of vascular fibrous tissue. Epithelial cells are fully differentiated. (X 198)

Fig. 15.—Hyperkeratosis of Larynx.

CASE 2.—White male, age 58. Film No. 63-20 (22-*f*18-3/4L).

History: Hoarseness for 5-6 weeks. Asthma for 5 years. Hemoptysis 2 years ago and occasionally since that time. Productive cough. Had constant sinusitis, especially on right side.

Histological Diagnosis: Hyperkeratosis of laryngeal mucosa.

Microscopic Description: Mass of markedly thickened and hornified stratified squamous epithelium with only a narrow zone of supporting stroma. The amount of hornification is considerable. There are no appreciable inflammatory exudates and there are no cancerous tissues. (X 198)

Fig. 16.—Pachydermia Laryngis.

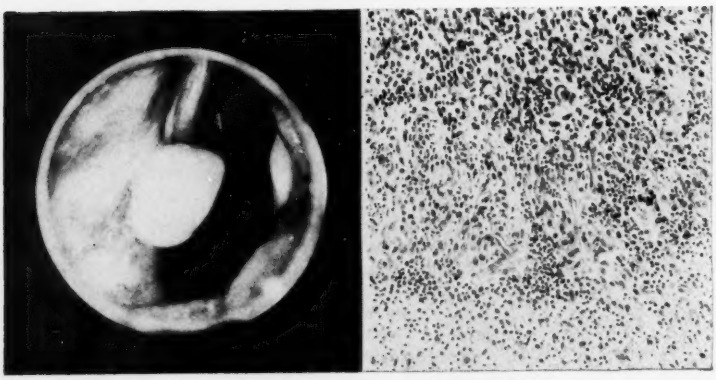
CASE 3.—White male, age 50. Film No. 63-1 (22-*f*15-3/4L).

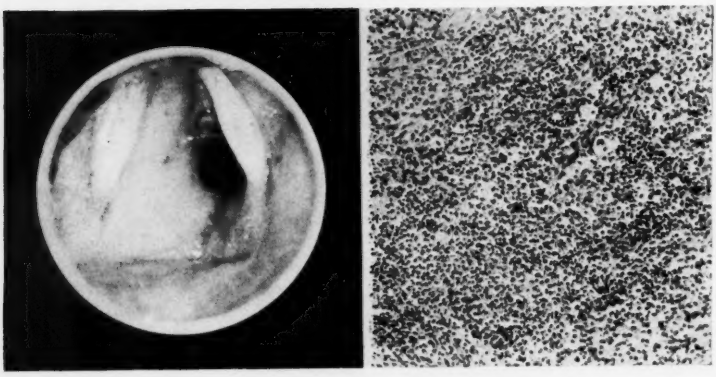
History: Pansinusitis and pharyngitis intermittently for many years. Purulent postnasal discharge. Ulceration and crusting of posterior pharyngeal wall. Intermittent hoarseness.

Histological Diagnosis: Hyperkeratosis of laryngeal mucosa.

Microscopic Description: Tissue has squamous cell epithelial margin, portions with wide surface layer of hornified cells. The deeper portions are fibrous tissue and skeletal muscle. The crevices along lining edge are also lined by thick layers of squamous epithelium. (X 198)

*The first number within the parentheses indicates the size of the General Electric flash lamp; the second number preceded by the letter *f* indicates the *f* stop; and the last number indicates the photographic laryngoscope used.





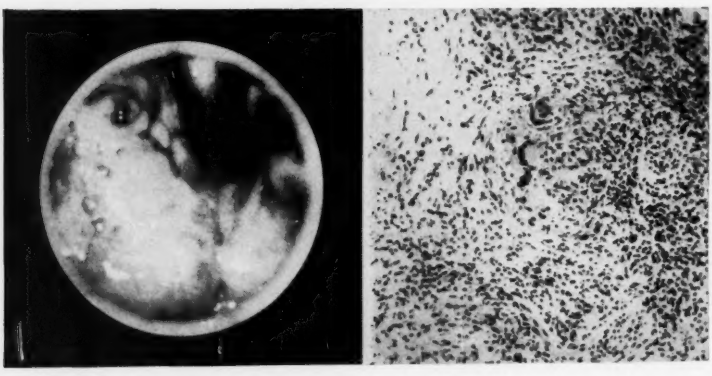


Fig. 17.—Nonspecific Granuloma of the Left False Cord Associated with Contact Ulcer.

CASE 4.—White male, age 54. Film No. 66-5 (22-*f*18-3/4L).

History: Hoarseness for 6 weeks becoming much worse suddenly.

Histological Diagnosis: Benign granuloma, left vocal cord.

Microscopic Description: Mass of vascular partially organized chronic granulation tissue. It has many small capillaries, and along the surface edge is a margin of fibrin also with exudate cells and some mononuclear phagocytes. There are no specific features in the chronic granulation tissues. (X 198)

Fig. 18.—Nonspecific Granuloma of the Larynx Producing Subglottic Stenosis.

CASE 5.—White female, age 34. Film No. 63-11 (22-*f*15-3/4L).

History: Multiple systemic infections of undetermined origin. Severe sinusitis; treatment elsewhere with sulfonamides, penicillin and streptomycin, with no results. Marked swelling and destruction of the nasal bridge with complete obstruction. Chronic infection of both ears necessitating a mastoidectomy. Tracheotomy 3 months ago because of bilateral subglottic edema. Negative serological and sputum examinations.

Histological Diagnosis: Chronic granulomatous inflammation.

Microscopic Description: Tissue lined by stratified squamous epithelium overlying tissue densely infiltrated by polymorphs, round cells and collections of large cells with abundant vacuolated cytoplasm and round vesicular nuclei. A few multinucleated giant cells are present and are similar to the large cells previously described. There is no evidence of sarcoidosis or tuberculosis. (X 198)

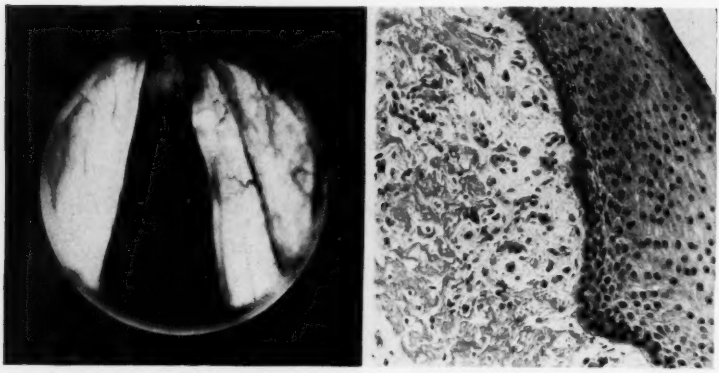
Fig. 19.—Laryngeal Tuberculosis Involving the Left Pyriform Sinus and Posterior Portion of the Larynx.

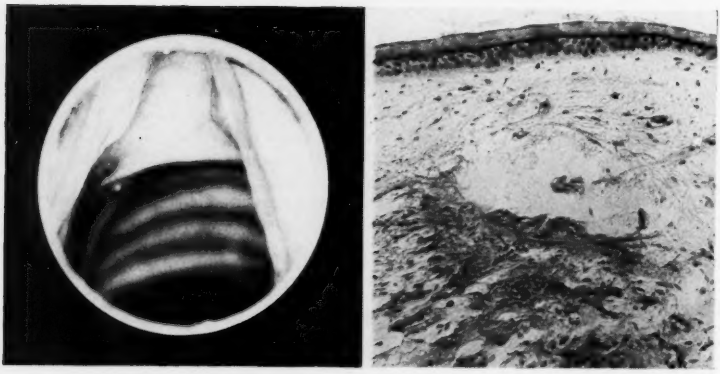
CASE 6.—White male, age 70. Film No. 64-9 (22-*f*15-3/4L).

History: Severe dysphagia for 3 weeks and a lump under the jaw which caused pain on swallowing sufficient to awaken the patient.

Histological Diagnosis: Chronic ulcerative tuberculosis (histologic), laryngitis.

Microscopic Description: Histologic preparations have along one margin a stratified squamous epithelium. Beneath, at some levels, are fibrous tissue thickenings, with dilated blood channels and focally along the lining edge in an edematous fibroblastic stroma are collections of lymphocytes, plasma cells and epitheloid cells. Other fragments have small structures, histological tubercles. In these are regions of caseations surrounded by epitheloid cells and with numerous Langhans's giant cells. At some levels are mucous glands. (X 198)





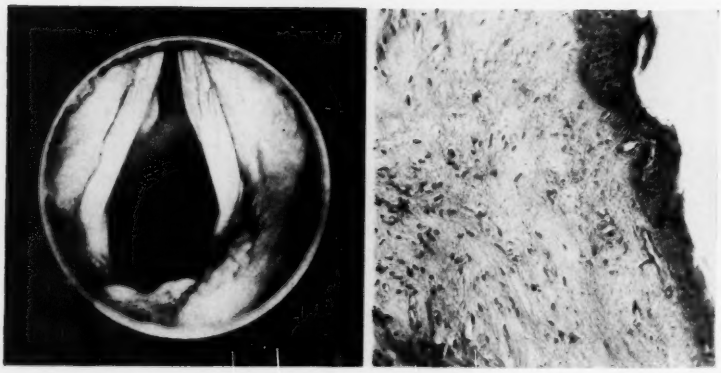


Fig. 20.—Mucous Retention Cyst in Anterior Third of the Right Vocal Cord.

CASE 7.—White male, age 58. Film No. 66-27 (22-*f*18-5/8L).

History: Hoarseness for 3 months.

Histological Diagnosis: Edema and fibrinoid degeneration of laryngeal mucosa.

Microscopic Description: Broad surface stratified squamous epithelium and a fibrous stroma with edema and fibrinoid changes. At some levels are small infiltrations of polynuclear leukocytes, lymphocytes, and mononuclear phagocytes. The surface stratified squamous epithelium is regular. (X 198)

Fig. 21.—Polyp in the Anterior Third of the Right Vocal Cord and Small Subglottic Web.

CASE 8.—White male, age 42. Film No. 60-1 (22-*f*15-3/4L).

History: Pain in throat which started 3 years ago. Silence for 6 months made "blood tumor" disappear and left white spot on cord. Had to talk over loud noises at work.

Histological Diagnosis: Edema and fibrinoid changes of laryngeal mucosa.

Microscopic Description: Narrow surface squamous epithelium and a fibrous stroma that is markedly edematous and has deposits of fibrinoid material. There are also some recent hemorrhages and at various levels deposits of brown coarsely granular material like old blood. Some small pieces of tissue are less markedly changed. The stroma tissues in these are more compact although edematous. (X 198)

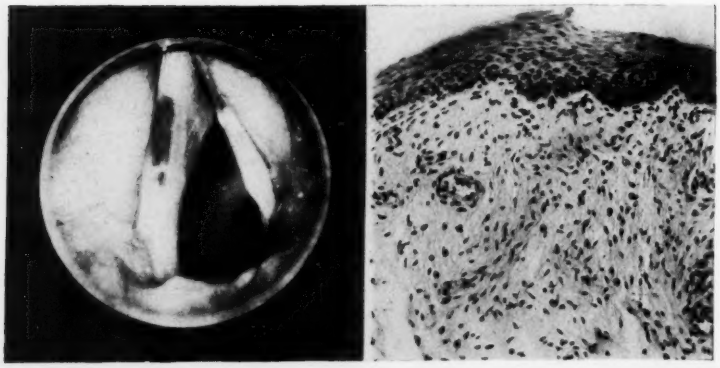
Fig. 22.—Polyp Below the Phonating Edge of the Left Vocal Cord at Junction of the Anterior and Middle Thirds.

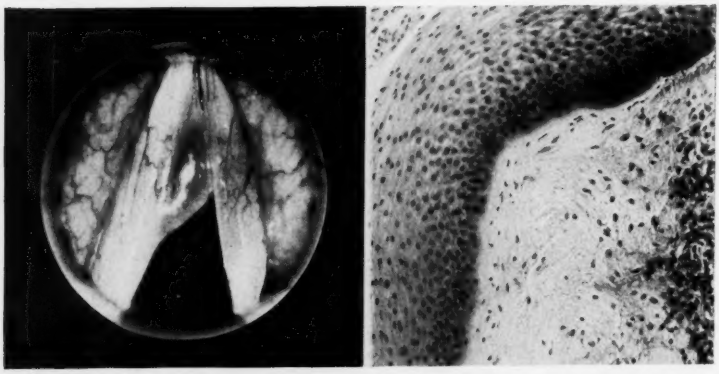
CASE 9.—White male, age 60. Film No. 66-30 (22-*f*15-3/4L).

History: Constant hoarseness for about 8 months, worse on arising, and improving during the day. No weight loss.

Histological Diagnosis: Edematous fold of laryngeal mucosa.

Microscopic Description: Markedly edematous fibrous stroma with some basophilic material in its interstices and with some fibrin deposits. Has a narrow regular surface squamous epithelial edge. There are no atypical stroma or epithelial structures. (X 198)





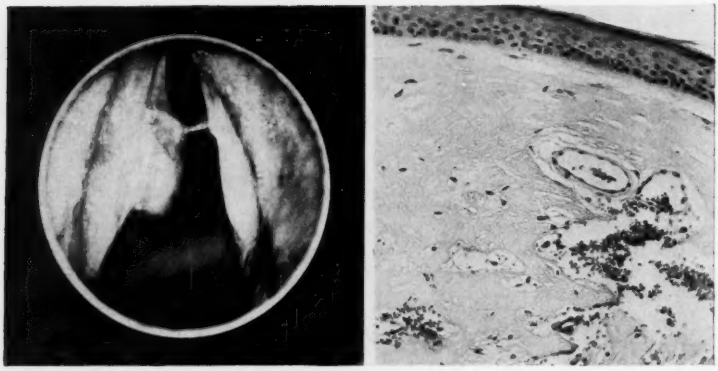


Fig. 23.—Polypoid Degeneration of the Left Vocal Cord and Hemorrhage Into the Superior Surface of the Cord.

CASE 10.—White female, age 48. Film No. 66-1 (22-*f*18-3/4L).

History: Constant hoarseness for one year.

Histological Diagnosis: Edematous mucosal tissues of the larynx.

Microscopic Description: Narrow squamous epithelial edge and a fibrous stroma, some portions with considerable edema. Along the squamous epithelial edge are foci of chronic inflammation. At one place is a tissue crevice lined by flattened epithelium and containing granular precipitates, some of them in oval concentrically arranged masses. There are no atypical stroma or epithelial tissues. (X 198)

Fig. 24.—Polyp of Anterior and Middle Thirds of the Left Vocal Cord.

CASE 11.—White female, age 60. Film No. 66-12 (22-*f*22-5/8L).

History: Persistent hoarseness.

Histological Diagnosis: Marked edema and exudative inflammation of the laryngeal mucosa.

Microscopic Description: Regular surface squamous epithelial edge. The fibrous stroma is edematous and has at some levels fibrinoid changes and at other levels an exudative inflammation and growth of fibroblastic tissues. At some levels there is considerable growth of fibroblastic tissues, and this is continuous with a mass of fibrin and polynuclear leucocytes. There are dense exudates of polynuclear leucocytes. Sections from various levels are without cancerous tissues. (X 198)

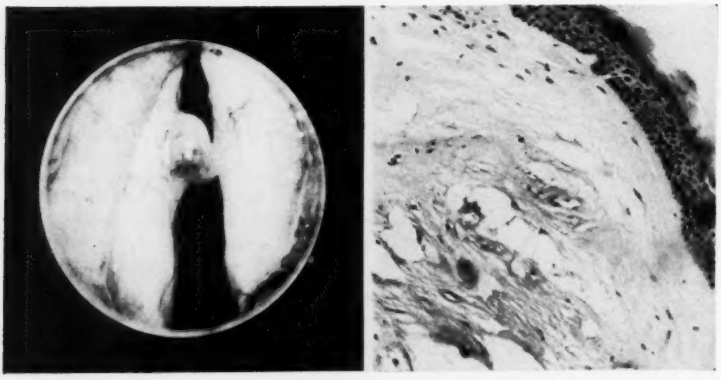
Fig. 25.—Polyp of Middle Third of the Left Vocal Cord with Strand of Mucus Across Glottis.

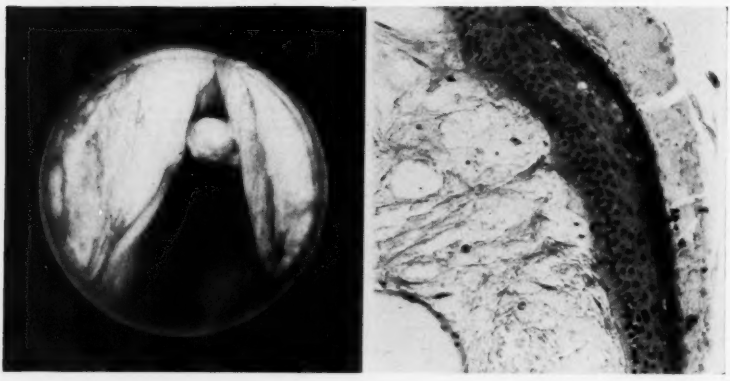
CASE 12.—White male, age 51. Film No. 66-36 (22-*f*15-3/4L).

History: Progressive hoarseness for 4-5 years. Productive morning cough progressively worse for 3 years. Gradually progressive dyspnea for 4 years.

Histological Diagnosis: Myxomatous polyp of vocal cord.

Microscopic Description: Tissue lined by stratified squamous epithelium which overlies a stroma whose connective tissue is markedly hyalinized. Patchy hemorrhages present. Occasional fragments of epidermis are acanthotic. Within the stroma are areas of edema with marked separation of fibrous tissues. (X 198)





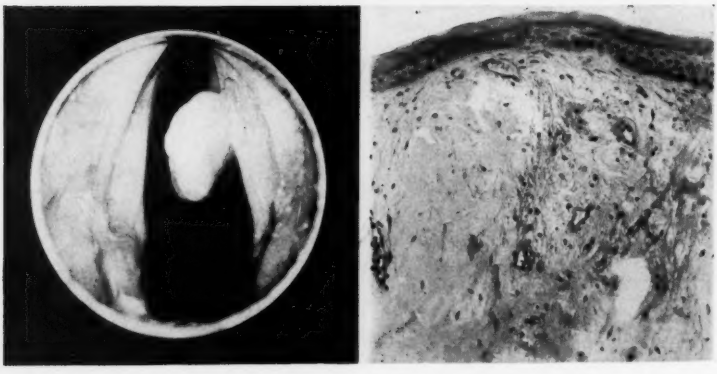


Fig. 26.—Polyp of Anterior and Middle Thirds of the Left Vocal Cord.

CASE 13.—White male, age 43. Film No. 66-19 (22-*f*15-3/4L).

History: Hoarseness and cough for 3 weeks.

Histological Diagnosis: Edematous fold of laryngeal mucosa.

Microscopic Description: Regular surface squamous epithelial edge extends along the margin of these tissues. The lining portions are edematous stroma with some tissue crevices, and portions of the stroma have undergone mucoid changes and have deposits of fibrin. (X 198)

Fig. 27.—Pedunculated Polyp of the Right Vocal Cord Arising at Junction of Anterior and Middle Thirds.

CASE 14.—White male, age 54. Film No. 66-10 (22-*f*18-5/8L).

History: Constant hoarseness for about 9 weeks, made worse by drinking liquids.

Histological Diagnosis: Edematous fold of laryngeal mucosa.

Microscopic Description: Narrow surface squamous epithelial edge and an edematous fibrous stroma with dilated blood channels. In this stroma are extruded red blood cells and deposits of fibrin. Along the surface edge is a little hornified epidermis. (X 198)

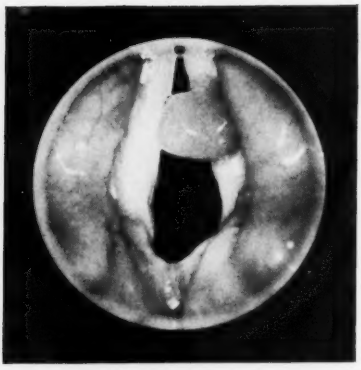
Fig. 28.—Pedunculated Polyp of Anterior and Middle Thirds of the Right Vocal Cord.

CASE 15.—White male, age 47. Film No. 66-39 (22-*f*18-3/4L).

History: Hoarseness for 1½ years; friend states it had been 3-4 years. Hoarseness increased in past few months. No pain.

Histological Diagnosis: Edematous fold of laryngeal mucosa.

Microscopic Description: Narrow surface squamous epithelial edge and a markedly edematous fibrous stroma with large tissue crevices and with fibrinoid changes. There is slight chronic inflammation or slight organization of the stroma tissues. (X 198)



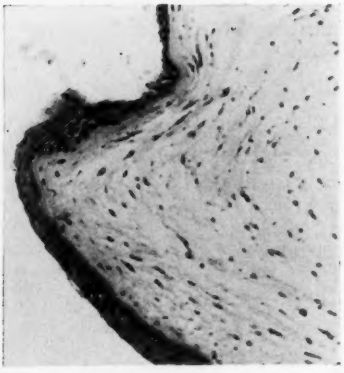


Fig. 29.—Pedunculated Freely Movable Polyp of the Right Vocal Cord. Subglottic Position During Inspiration Showing Attachment to the Subglottic Region of the Vocal Cord.

CASE 16.—White female, age 32. Film No. 66-34 (22-*f*15-3/4L).

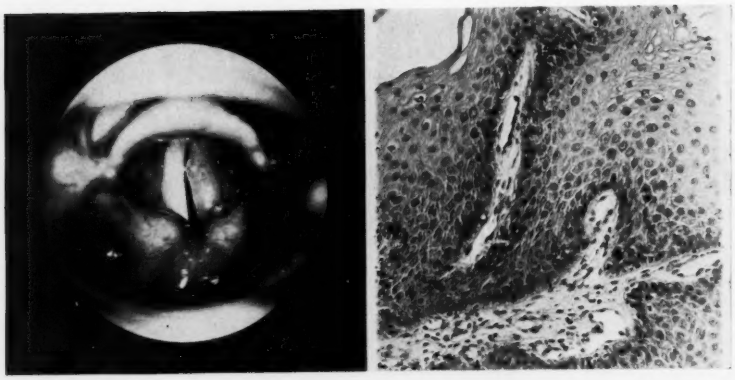
History: Continuous hoarseness for past 5 years. No change during this time. No hemoptysis, sweats or cough.

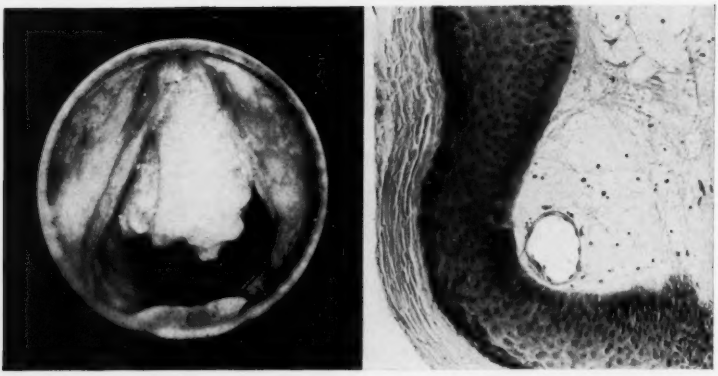
Histological Diagnosis: Myxomatous polyp of vocal cord.

Microscopic Description: Polypoid structure lined by stratified squamous epithelium which overlies loose fibrous stroma. Within the latter, there are hugely dilated partly thrombosed blood vessels. (X 198)

Fig. 30.—Supraglottic position during expiration. Film No. 66-35 (22-*f*15-3/4)L).

Fig. 31.—Photomicrograph of polyp.





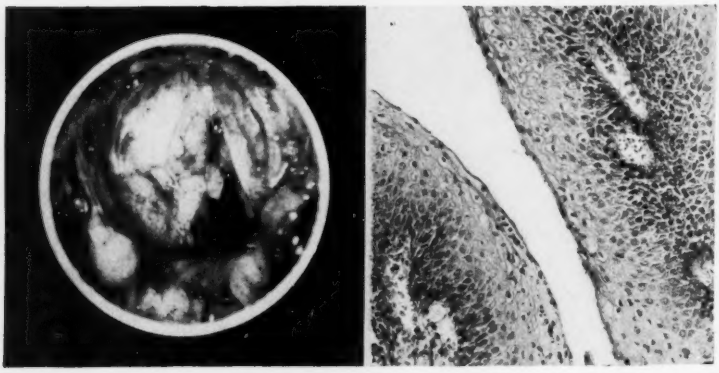


Fig. 32.—Papilloma of Right Vocal Cord, Indirect View.

CASE 17.—White female, age 20. Film No. 56-2.

History: Marked hoarseness since awakening about 2 weeks ago. Had some associated dyspnea.

Histological Diagnosis: Papilloma of larynx.

Microscopic Description: Very marked hyperplasia of squamous epithelium in a typical papilloma arrangement. These cells appear highly pyknotic but show no evidence of anaplasia. There is a hornifying layer of desquamated epithelium on all sides of the peripheral border of the tissue. (X 198)

Fig. 33.—Large Papilloma Arising From the Right Vocal Cord, Anterior Commissure, and Anterior Portion of the Left Vocal Cord.

CASE 18.—White female, age 48. Film No. 66-21 (50-*f*18-3/4L).

History: Hoarseness for many months; increasing dyspnea for several weeks.

Histological Diagnosis: Papilloma of the larynx.

Microscopic Description: These tissues have the structure of a papilloma of the larynx. Stalks of edematous fibrous tissue are covered by a stratified squamous epithelium. This varies somewhat in width, but generally is wide. (X 198)

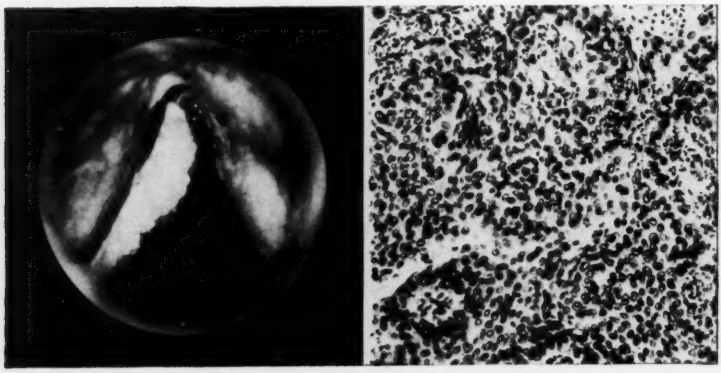
Fig. 34.—Friable Freely Bleeding Papilloma in a Small Child Involving the Entire Larynx.

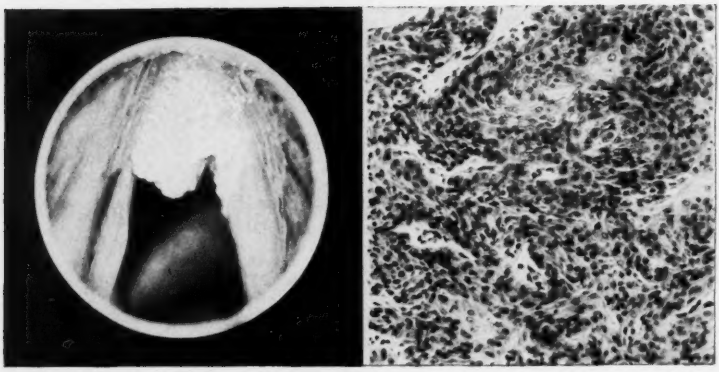
CASE 19.—White male, age 3. Film No. 66-26 (50-*f*22-3/4L).

History: Aphonia, dyspnea, and tracheotomy 1½ years ago.

Histological Diagnosis: Papilloma of larynx.

Microscopic Description: Small fragments are of papillomatous character covered by a thick hyperplastic, stratified squamous epithelial layer permeated by polymorphonuclears. Papillae and base are vascular, somewhat edematous with diffuse but moderately light round cell infiltration. No evidence of malignancy. (X 198)





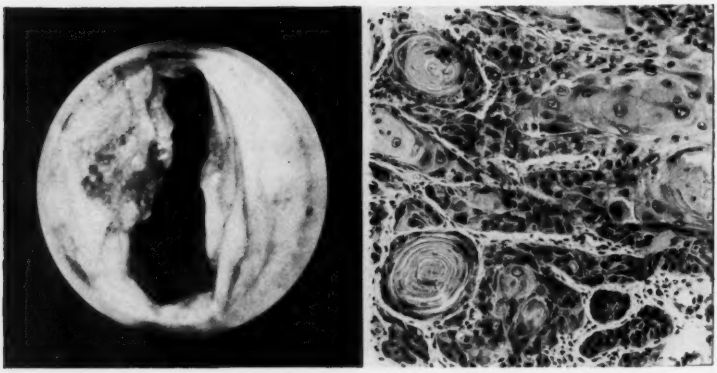


Fig. 35.—Carcinoma Involving the Anterior Two Thirds of the Vocal Cord.

CASE 20.—White male, age 56. Film No. 67-1 (22-*f*15-5/8L).

History: Hoarseness for 6-8 weeks. Did not clear completely, although it improved at times.

Histological Diagnosis: Stratified squamous cell carcinoma of larynx.

Microscopic Description: These pieces of tissue have a fibrous stroma that is extensively ingrown by large and small irregular masses of stratified squamous epithelium. The cells repeat the middle and surface layers. There is little hornification and among the cells are some in mitosis. (X 198)

Therapy: Laryngofissure.

Fig. 36.—Carcinoma of the Right Vocal Cord, Anterior Commissure, and Anterior Portion of the Left Cord.

CASE 21.—White male, age 73. Film No. 67-34 (50-*f*18-3/4L).

History: Sensation of dryness in throat for 2 years. Hoarseness for 6-8 months and cough for 3 months. No pain or weight loss, but stated he thinks he may have a little bronchitis.

Histological Diagnosis: Stratified squamous cell carcinoma of the larynx.

Microscopic Description: These tissues have only scanty fibrous stroma with inflammatory exudates. They are mainly large and small irregular masses of ingrown stratified squamous epithelium. The cells are arranged in mosaics and repeat the middle and surface layers of squamous epithelium. There is considerable hornification in the larger masses, occasionally in the form of pears. (X 198)

Therapy: Laryngectomy.

Fig. 37.—Carcinoma Involving the Left Side of the Larynx and Anterior Commissure.

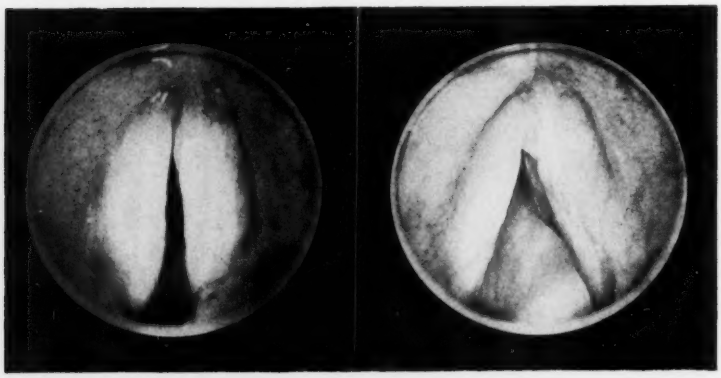
CASE 22.—White male, age 68. Film No. 67-27 (50-*f*18-5/8L).

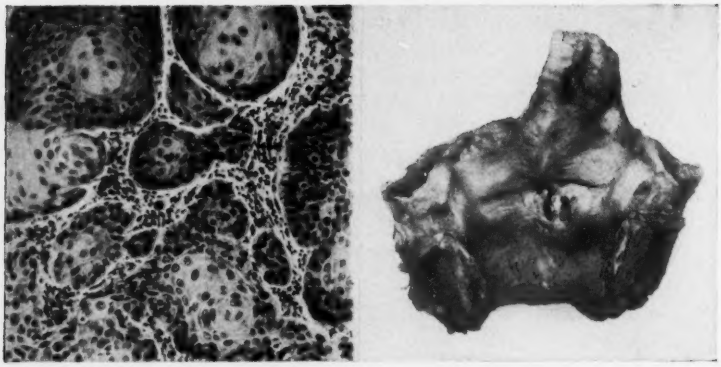
History: Hoarseness for 3 years. During the last month, the hoarseness had increased and he had noticed some dyspnea on exertion. Otherwise his health was excellent.

Histological Diagnosis: Stratified squamous cell carcinoma of the larynx.

Microscopic Description: These tissues have along one edge a surface squamous epithelium with a wide edge of hornified epidermis. Between is a fibrous stroma, rather scanty in amount, with large and small irregular masses of ingrown stratified squamous epithelium. There is considerable hornification of these masses. Many of the large and also of the small cell aggregates have centers with concentrically arranged hornified material. (X 198)

Therapy: Laryngectomy.





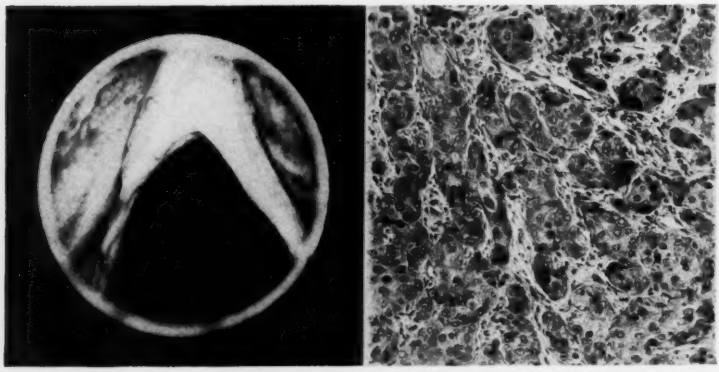


Fig. 38.—Subglottic Carcinoma of the Right Vocal Cord During Expiration and Inspiration, Revealing the Tumor Only During Inspiration.

CASE 23.—White male, age 50. Film No. 67-20 (22-*f*12-3/4L) and 67-22 (22-*f*15-3/4L).

History: Thoracoplasty and subsequent arrest of pulmonary tuberculosis 5 years previously. Persistent hoarseness for six months.

Histological Diagnosis: Stratified squamous cell carcinoma of larynx.

Microscopic Description: Histological preparations are mainly large and small masses of stratified squamous epithelium. The large ones contain masses of hornified cells, the small masses have ingrown into a fibroblastic stroma with inflammatory exudate. They also simulate the surface layer of squamous epithelium. (X 198)

Therapy: Laryngectomy.

This case illustrates the necessity for observation of the larynx during complete cycles of phonation and respiration. During expiration and phonation the tumor is not visible but during inspiration or when the cord is retracted with the lip of the laryngoscope, the tumor is visible in the right subglottic area.

Fig. 39.—Photomicrograph of biopsy specimen, Case 23.

Fig. 40.—Laryngectomy specimen, Case 23.

Fig. 41.—Larynx Nine Months After Anterior Commissure Laryngofissure of Left Vocal Cord. Photomicrograph of Biopsy Specimen.

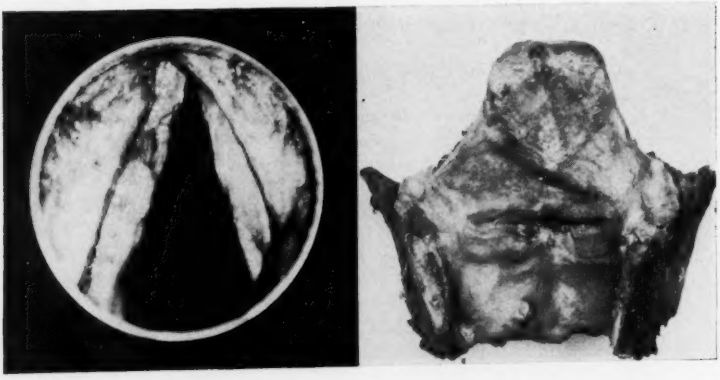
CASE 24.—White male, age 58. Film No. 67-13 (22-*f*15-3/4L).

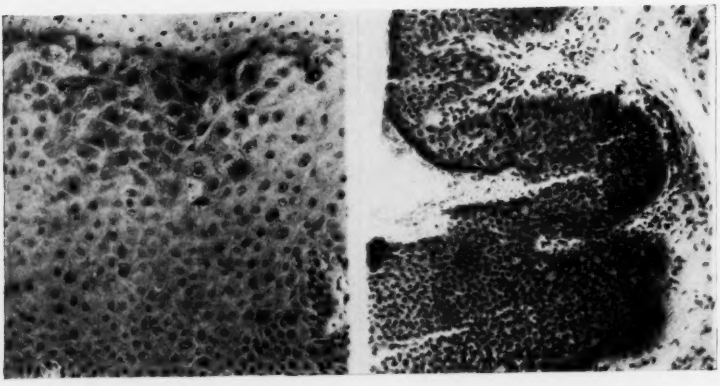
History: Hoarseness for 6 months of varying degree. No pain.

Histological Diagnosis: Stratified squamous cell carcinoma of larynx.

Microscopic Description: These pieces of tissue have a scanty fibroblastic stroma with inflammatory exudates and are extensively ingrown by many large and small masses of stratified squamous epithelium. The centers of the large masses are hornified. (X 198)

Therapy: Laryngofissure.





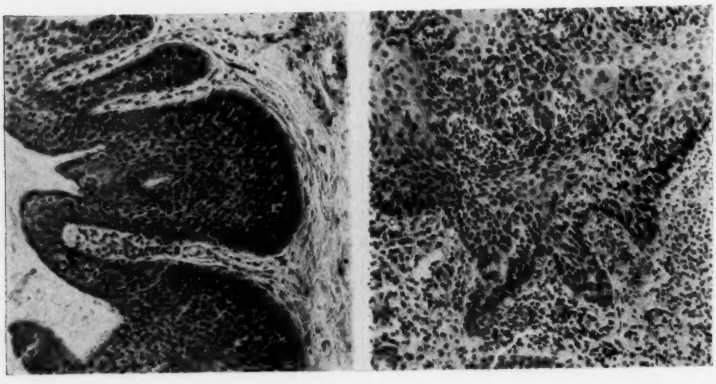


Fig. 42.—Carcinoma of the Left Vocal Cord with Subglottic Extension: Direct Laryngoscopic View and the Laryngectomy Specimen.

CASE 25.—White male, age 58. Film No. 67-10 (22-*f*18-3/4L).

History: Hoarseness at various times following colds for the last 8-12 months. General weight loss about 30 pounds in the last 6-10 years. Last attack of hoarseness which came on suddenly after a cold was associated with a productive cough and marked shortness of breath. Hoarseness persisted after direct laryngoscopies and conservative treatment.

Histological Diagnosis: Final: Oct. 12, 1946. Squamous cell carcinoma of larynx.

Microscopic Descriptions: Final: Oct. 12, 1946, Fig. 46. Tissues show a broad squamous epithelial edge and a little stroma with inflammatory exudates. The squamous cells in this broad layer are arranged in mosaics. They repeat the middle and surface layers. Among them are some in mitosis, and the appearance of some of these cells approaches the atypical. Among the cells are some concentrically arranged massed with hornification. The stroma beneath is fibroblastic with inflammatory exudates. Along the surface edge is a zone with hornification. Although the histologic preparations again do not reveal deeply invasive properties in the sense of growth of cell masses deeply into the stroma, the atypical structures of the cells, the fact that this epithelium, clinically, has shown growth potentials by recurrence of new tissues, leaves the impression that this is squamous cell carcinoma, but superficial in extent. (X 198)

Therapy: Laryngectomy.

This case illustrates the progressive nature of a laryngeal lesion. Over a period of 1 year and 10 months repeated biopsies revealed benign lesions, and it was not until the fifth biopsy that carcinomatous tissue was obtained. This illustrates the necessity for close observation and repeated biopsy of suspicious laryngeal lesions.

Fig. 43.—Photomicrograph of 1st biopsy specimen (Jan. 27, 1945). Hyperkeratosis of laryngeal mucosa. 2nd biopsy (not shown) revealed: Edematous and hemorrhagic laryngeal mucosa.

Fig. 44.—Photomicrograph of 3rd biopsy specimen (May 26, 1945). Chronic inflammation and slight squamous epithelial thickening of laryngeal mucosa.

Fig. 45.—Photomicrograph of 4th biopsy specimen (Oct. 5, 1946). Papillary hyperplasia of larynx.

Fig. 46.—Photomicrograph of 5th and last biopsy specimen (Oct. 12, 1946). Squamous cell carcinoma of larynx.

LIV

THE PRESENT STATUS OF THE DIAGNOSIS AND TREATMENT OF ENDOLYMPHATIC HYDROPS (MÉNIÈRE'S DISEASE)

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ROCHESTER, MINN.

In 1861, Ménière[1] described a syndrome of paroxysmal attacks of nausea and vomiting with progressive deafness and pounding tinnitus in the affected ear. He described in detail most of the characteristic symptoms and signs of the malady that came to be called by his name: the sudden onset of the attacks without any observable relation to previous illness or precipitating motion of the head or body; the pounding tinnitus with progressive loss of hearing, which may have preceded the first attack of vertigo by many years, in one and occasionally in both ears. He noted the tendency for the tinnitus to be increased during the attack. He stated that the impairment of hearing might progress to complete deafness without any gross pathologic findings suggestive of disease in the ear.

Unfortunately Ménière presented as a probable basis for his syndrome the pathologic findings in the case of a young girl, who during her menses, after riding all night on the top of a diligence exposed to the cold of winter, experienced continuous vertigo which was increased on movement of the head; complete deafness in both ears; severe nausea and vomiting on movement of the head. Death occurred on the fifth day of the illness. At necropsy the gross pathologic findings were stated to be bloody exudate in the semicircular canals and traces of exudate in the vestibule but none in the cochlea.

M'Kenzie[2] pointed out that, whatever the patient had, it obviously was not Ménière's disease. He discovered after a prolonged search that a pathologic report on the same patient had been presented by Ménière 14 years before his paper of 1861. In the first report Ménière stated that the entire labyrinth had been filled with bloody exudate and no mention was made of vertigo. M'Kenzie felt

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The Charles Raymond Hume Memorial Address; read before the meeting of the Louisiana-Mississippi Ophthalmological and Otolaryngological Society, Biloxi, Miss., May 5, 1947.

that this pathologic report of Ménière was an outstanding example of careless reporting and faulty thinking but that great credit was due Ménière as the first physician to put to clinical use the new knowledge of labyrinthine physiology brought out by Ewald.[3] Previously attacks such as Ménière described had been attributed to "apoplexy." This pathologic description, however, was further garbled in subsequent quotation into "hemorrhage into the labyrinth" and resulted in much confusion as to the symptoms and pathologic changes of Ménière's disease. Finally, any condition in which either dizziness and giddiness or vertigo was present was included in a so-called Ménière's symptom complex or syndrome without regard to the original clinical description of Ménière.

Crowe,[4] however, pointed out that the actual clinical condition originally described by Ménière is sufficiently characteristic to justify its attribution to some specific type of labyrinthine disorder and insisted that the term, "Ménière's disease," rather than "Ménière's syndrome," ought to be retained. As a matter of fact, little of importance has been added to the original clinical description of Ménière.

Mygind and Dederding[5-7] pointed out the fluctuating character of the deafness in Ménière's disease and noted the frequent occurrence of sudden, severe headaches, apparently of a vasodilating type, especially in the intervals between attacks of vertigo, and the frequent association of Ménière's disease with Quincke's edema, urticaria and other so-called allergic (or pathergic) conditions.

Crowe also emphasized the fluctuating character of the deafness in Ménière's disease and gave the following reasons among others for suspecting that the origin of the symptoms is in the labyrinth: the vestibular symptoms differ only in intensity from those produced by functional tests; the infrequency of a vestibular symptom arising from intracranial tumors argues against the disease being in the nerve or the vestibular nuclei; involvement of other cranial nerves is absent in Ménière's disease; choked disk is not present; trauma of the vestibular nerve does not cause symptoms. Crowe accurately foresaw the eventual pathologic picture of Ménière's disease when he stated that the strongest reasons for suspecting variable pressure and chemical changes in the membranous labyrinth as the cause of Ménière's disease are the frequency and length of the remissions.

Brunner[8] stated that labyrinthine vertigo which reaches a high degree of intensity is always accompanied by nystagmus of the third degree of intensity and the quick component is always directed to-

ward the normal ear. He stated that patients who had hysteria might complain of severe dizziness but spontaneous nystagmus was never present.

Lindsay[9] found three patients with Ménière's disease who had positional vertigo in the intervals between attacks. He felt that from the standpoint of the history positional vertigo in the intervals of attacks was relatively common. Shambaugh[10] described the "diplacusis" that occurs between attacks in cases of unilateral Ménière's disease. In diplacusis a pure tone is heard in the affected ear as being higher in pitch than in the uninvolved ear. Shambaugh interpreted this to mean that the vibrating membrane in the involved ear was weighted by edema so that it would move less readily and consequently refer vibrations closer to the basal coil.

Wright[11, 12] stated that the types of dizziness arising from the Ménière type of labyrinthine derangement are: (1) typical semicircular canal attacks in which the patient has the feeling that objects are rotating around him or that he himself is rotating; (2) a sensation of being forced to one side or the other, which sensation may be actually accompanied by an inability to walk straight; (3) attacks in which the patient is suddenly thrown to the ground either forward or backward as if he had been hit on the head with a hammer (possibly due to stimulation of the otolith organs); (4) occurrence of a more or less constant unsteadiness so that the patient has to hold on to a support and both feels and is unsteady.

There is a confusion in the minds of many commentators on Ménière's disease or "syndrome" between the terms "dizziness" or "giddiness" and "vertigo." Some of the confusion in regard to the proper differential diagnosis between Ménière's disease and other conditions in which either vertigo or dizziness is present would be corrected by a more careful use of terms. Weiss[13] stated that because patients describe vertigo or dizziness indiscriminately as "swimming," "reeling," "giddiness," "lightheadedness," "faintness," "drunk feeling" or a sense of "foolishness" the first step in the treatment of vertigo or dizziness is the proper interpretation of the patients' complaints. Without this, effective treatment is not possible. Weiss defined vertigo in the strict sense as an experience in which the patient has the sensation that the outer world is moving about him (objective vertigo—eyes open) or that he himself is whirling in space (subjective vertigo—eyes closed). Frequently, however, the term vertigo is used erroneously as a synonym for dizziness or giddiness to indicate a disturbed relationship to surrounding objects in

space. The terms dizziness and giddiness should be restricted to an abnormal sensation of unsteadiness characterized by a feeling of movement within the head, without the sensation of the external world or the patient himself being in motion. Dizziness or giddiness as a subjective sensation should not be confused on the other hand with the sensation of unsteadiness associated with ataxia. At times true vertigo and dizziness can coexist.

Vertigo is the result of certain types of disturbance of the nervous mechanism involved in the maintenance of normal body balance. While the master organs in maintaining balance are the vestibular portions of the labyrinth with their neural connections through the vestibular portion of the eighth cranial nerve to the vestibular nuclei in the medulla, important contributory roles are played by the ocular muscles, the muscles of the head and neck, proprioceptive sensations from the muscles and tendons in general and exteroceptive sensations from the skin and retinas of the eyes.

The hair cells of the cristae ampullares and maculae of the utricle and saccule send out neuraxons which unite to form the vestibular portion of the eighth cranial nerve. The cell bodies of the vestibular fibers lie in Scarpa's ganglion. According to McNally[14] the saccule does not subserve any equilibratory function. According to Rasmussen[15] the eighth cranial nerve ends on the dorsal side of the pons in three vestibular nuclei from which relay fibers follow three pathways. The ascending fibers of the first pathway join the medial longitudinal fasciculi of the same and opposite sides and pass to the nuclei of the third, fourth and sixth cranial nerves. Through these connections reflex movements of the eyes are brought about. The descending fibers of the second pathway constitute the vestibulospinal tract. Through these connections impulses are conveyed to the skeletal muscles. The fibers of the third pathway pass to the cerebellar nuclei by way of the inferior cerebellar peduncles. The vestibular nuclei are thought to be connected with the nucleus of the vagus nerve and through it with the autonomic nervous system; hence pallor, sweating, nausea and vomiting are associated with severe nystagmus. Impulses conveyed along the direct sensory cerebellar tract and thence through the superior cerebellar peduncle to the cerebrum are believed to give rise to the sensation of vertigo.

Nickle[16] stated that in normal states this interlocking control mechanism, which is mostly bilaterally represented, is so poised that there is compensation for all movements and changes of position of any part of the body. If any part of this system gets out of step

with the system as a whole, however, that relationship is upset and conflicting impressions are transmitted which the higher reflex centers cannot coordinate, the resultant confusion becoming either dizziness, vertigo or unsteadiness. True vertigo arises from disturbances of the vestibular end-organ or possibly of the vestibular nuclei. Dizziness and unsteadiness (incoordination) may arise from disturbances of the proprioceptive, exteroceptive or central portion of the mechanism.

When impulses arising in the vestibular nerve at its origin in the labyrinth produce undue stimulation, vertigo and nystagmus result. This fact is made use of in making functional vestibular tests. Stimulation of the labyrinth itself seems to produce a much more marked sense of angular acceleration about an axis or linear acceleration than irritation arising in the vestibular nuclei. The most important difference between vertigo due to labyrinthine involvement and vertigo due to lesions of the brain stem, according to Alpers,[17] lies in the clear-cut nature of vertigo associated with peripheral disease. In vertigo associated with central lesions, patients find it difficult even with help to define precisely the nature of their sensation. Patients who have labyrinthine vertigo have no such difficulty. Central vertigo (sometimes mere dizziness) may begin when the patient is at rest and it is independent of movement. This distinguishes it from dizziness or unsteadiness produced by disturbances of the proprioceptive and exteroceptive sensations other than increased pressure in the endolymph.

Kobrak[18] has raised the question whether vertigo and nystagmus are parallel phenomena or whether vertigo is secondary to central stimulation produced by the nystagmus. In his opinion the latter is true. Best and Taylor[19] pointed out that the vertigo produced by turning tests differs from nystagmus in that nystagmus is always in the plane of rotation, whereas vertigo after rotation may vary in its direction depending on changes in the position of the head. The reason for this difference is that, whereas nystagmus is reflex, the sensations of vertigo are largely the result of cerebral interpretation of movement of endolymph and the position of the head at the moment is taken into account. This appears to reinforce the opinion of Kobrak. According to Leidler,[20] however, there is dissociation of vertigo and other vestibular symptoms in cases of nonsuppurative encephalitis.

Tait[21] pointed out that nystagmus is a double affair. The eye first rotates slowly in one direction and then by a sudden jerk or flick rotates in the opposite direction, a sequence of events which

may go on for a time in succession. Arellano[22] explained that the purpose of the slow component is to hold the existing visual panorama steady notwithstanding movement of the head. He has shown that during the flick stage visual cortical registration does not occur. The ocular reflexes involved in movement of the eyes as the result of labyrinthine stimulation are of two kinds according to Tait, depending on the receptors concerned in the stimulation: (1) nystagmic and (2) slow components without the flicklike jerks of nystagmus. The musculature involved in maintaining an adequate view of the surroundings, even though the pedestal or support of the registering retina be temporarily rocking or swinging, can be conveniently divided into two categories: (1) the ocular muscles and (2) other bodily musculature. It would appear that in nystagmus only proprioceptive sensations from the ocular muscles need be considered.

Hyndman[23] expressed the opinion that two independent reflex arcs are concerned with nystagmus and this is also the contention of Démétriades and Spiegel[24] as a result of their findings based on sectional operations on the brain stem. Hyndman felt that while the point of origin of both the quick and the slow phase of the nystagmus is in the vestibular nuclei, probably two distinct pathways lead thence to the motoneurons of the ocular muscles. Hyndman stated in substantiation of this hypothesis that voluntary deviation of the eyes and conjugate deviation associated with an epileptic seizure are not nystagmoid if the cerebellum is intact. To be clonic the reflex must be occasioned through subcortical levels.

Hyndman felt that in explaining the quick component of nystagmus a close analogy may be drawn to ankle clonus. If one is to obtain an extensor response (extensor being used in the physiologic sense), the extensor muscles of the ankle must be stretched. This stimulus is followed by involuntary contraction which, if allowed to occur, becomes the adequate stimulus for release. If the original stimulus—that is, the forceful dorsiflexion of the foot—is maintained, the response proceeds in the characteristic clonic rhythm. It is not too imaginative to see the same mechanism at work in nystagmus. The maintaining force here is the stimulus from the labyrinth which continues to impose an increased tone in the extraocular muscles involved. When the muscles have shortened sufficiently, the stimulus for release from contraction is given through the afferent connections from the proprioceptors in the muscles and the quick component follows. The labyrinthine stimulus for increased tone is still present, however, and the process assumes a rhythmic character. This hypothesis involves the supposition of

two reflex arcs, one subserving the contraction phase and the other the release phase. It appears that the latter is more cephalic than the former and can be eliminated as an independent reflex.

Nystagmus is apparently related only to stimuli arising from the vertical and horizontal canals. McNally[25] has shown that nystagmus never rises from utricular stimulation. Stimulation of the macula gave a slow deviation of the eyes in a compensatory direction but nystagmic action did not ensue. This observation might seem to cast doubt on the hypothesis presented by Hyndman but it is doubtful whether the movement produced by stimulation of the utricular macula is sufficient to stimulate the stretch reflex in the ocular muscles. It is obvious, however, that utricular stimulation causes impulses transmitted to the vagus and sympathetic nerves. Seasickness or motion sickness, which McNally points out is due to linear acceleration affecting the utricles, is not associated with nystagmus but is accompanied by nausea, pallor, cold sweat and vomiting.

It seems doubtful whether true subjective or objective vertigo occurs other than exceptionally from stimulation above the vestibular nuclei, although it has been reported. These reports seem to depend in the main on confusion as to the definition of vertigo. Foerster,[26] however, observed that stimulation of the superior lip of the interparietal sulcus evokes violent vertigo. The patient either sees the objects before him moving toward the side of the stimulation or has the sensation of turning to the opposite side. Central vertigo (strictly interpreted), however, appears to be a rare clinical symptom if psychogenic vertigo is excluded.

There has been some controversy as to how the stimulus of angular acceleration about an axis is transmitted to the hair cells of the crista of a horizontal canal. The work of Steinhausen[27] and of Dohlman[28, 29] seems to have settled the problem satisfactorily.

By injecting India ink into the membranous labyrinth of living fish and laboratory animals and freshly dead human cadavers, Steinhausen demonstrated that the cupula reached from the crista to the opposite wall of the vestibule and formed an endolymph-tight contact with it. Movement of the endolymph from rotation produced a bending of the cupula in the direction of its flow, the return of the cupula to its starting position being by virtue of its elasticity and its tendency to resist torsional stress.

Dohlman showed that after the deforming stress was removed it took about 30 seconds for the cupula to return to its starting po-

sition. He gave further evidence which showed that cooling of the endolymph increased its weight. With the canal to be tested placed in the vertical position, the increased weight of the endolymph produced displacement of the cupula. When the tested canal was placed horizontally, removing the gravitational stress, the elasticity of the cupola again returned it to its starting position in approximately 30 seconds, thus corresponding to the results obtained by rotation. Dohlman found that if the deformation of the cupula was maintained in the cold caloric test the length of the recovery period by the two methods of testing could not be compared and vertigo and nystagmus tended to be persistent. Dohlman also found that by trephining the skull of a rabbit and directing the cold water against the medial surface of the horizontal canal, thus reversing the direction of endolymphatic flow, he was able to reverse the direction of the nystagmus in the cold caloric test. This goes far to establish the hypothesis of a bidirectional receptivity of a single canal. Ewald[3] in the pigeon, McNally[30] in the frog and Steinhausen in the pike found that an ampullopetal flow of endolymph stimulates but that an ampullofugal movement of endolymph in the horizontal canal fails to stimulate. Thus it appears that in the higher vertebrates an additional complexity of propriation, as Tait stated, has arisen.

Regarding the localization of the pathologic changes causing central vertigo, Leidler differentiated general, lateral and caudal-oral forms of localization of lesions of the central vestibular system producing vertigo according to their relation to the midline of the fourth ventricle and the nucleus of the sixth cranial nerve. Lateral localization in man may be postulated on the basis of these facts: lesions involving the second neuron are usually accompanied by spontaneous, horizontal nystagmus to the focal side; lateral forced attitudes in attacks of vertigo speak for a focal point in the posterior longitudinal bundle on the side of the forced attitude. Leidler divided the caudal-oral extension of the central vestibular system into the parts lying posterior and anterior to the length of the nucleus of the sixth cranial nerve. In the posterior portion abnormal reactions were called forth by lesions in the substantia reticularis. The abnormality usually consists in an appreciable preponderance of the slow components of the horizontal nystagmus. The nearer the lesion approaches the apex of the sixth nucleus, the more does the predominance of the slow component become apparent until finally as the height of the nucleus is reached the quick component vanishes entirely and a single phase reaction occurs. If the lesion is located

orally from the sixth nucleus, single phase reactions of the eyes and head may occur spontaneously as well as through vestibular stimulation. Deviation of the gaze to one side, or especially upward, speaks for a focus in the height of the posterior commissure. In concurrence with the observation of Leidler, Lorente de Nó[31] traced the center for the quick component to the substantia reticularis.

Functional Tests of Diagnostic Significance in Endolymphatic Hydrops. Simonton[32] studied the vestibular reactions to the cold caloric test in 75 patients seen at the Mayo Clinic who had endolymphatic hydrops. He found the results disappointing in indicating the canal involved, the degree of involvement or the site of the lesion in unilateral involvement. He found evidence of bilaterality in 11 of the 75 patients studied.

This appears to have been the consensus of observers until Fitzgerald and Hallpike[33] described directional preponderance in caloric tests. Cawthorne, Fitzgerald and Hallpike[34] studied 50 cases by this method. With the patient lying supine with the head elevated 30 degrees from the horizontal, water at 7° C. above and below the body temperature is instilled for 40 seconds into the ear being examined. The measure of the response is taken as the time in seconds between the application of the stimulus and the end of the response. In explanation of directional preponderance Hallpike[35] stated that of the four reactions partaking in directional preponderance each ear contributes a particular disturbance of its own two reactions. Thus in the case of directional preponderance to the right, the normal relationship of the cold to the hot reaction of the left ear is altered in favor of the cold and that of the right ear in favor of the hot. If, for example, one labyrinth is defunct, the presence of a directional preponderance can still be correctly deduced from a characteristic alteration in the responses of the opposite ear. This is of importance inasmuch as directional preponderance may arise as a result of unilateral labyrinthine destruction. Contrary to Ewald's law, Hallpike found that the cold stimulus causing an ampullofugal flow of endolymph gives the larger response.

In discussing caloric tests Dohlman pointed out that in radical mastoidectomy cavities the labyrinth was responsive to changes of 0.5° C. and that the latent period in normal ears represented the time it took to bring the tissues surrounding the labyrinth to a temperature low enough to transmit an effective stimulus to the labyrinth. Therefore in caloric testing one was measuring the absorptive properties of the surrounding tissues for either hot or cold as well

as the functional condition of the labyrinth. Even though it had been pointed out that in the caloric test one can compare the reactivity of one ear with that of the other, there still seem to be too many uncontrolled variables in a caloric test to allow general conclusions to be drawn.

Cawthorne, Fitzgerald and Hallpike found that the directional preponderance test showed a vestibular lesion in 47 of 50 cases in which a clinical diagnosis of endolymphatic hydrops had been made. In 42 cases the test showed the vestibular lesion to be on the side of the ear in which hearing was worse. This would suggest that there is approximately a 16 per cent error in diagnosis by the method of testing. While this is superior to the result obtained by previous methods of testing, it would not seem to add much to a diagnosis obtained by a well-taken history and tests of cochlear function. From their findings Cawthorne and his associates regarded endolymphatic hydrops as essentially bilateral, some evidence of loss of cochlear function being found on both sides in 86 per cent of the 50 patients studied.

Pathology of Endolymphatic Hydrops. Fortunately much of the confusion and controversy in regard to Ménière's disease has been dissipated by the discovery of the true pathologic picture in authenticated cases of this disorder. Since the first two cases were reported by Hallpike and Cairns[36] 18 additional cases have been reported by Yamakawa,[37] by Hallpike and Wright,[38] by Rollin,[39] by Lindsay[40] and by Altmann and Fowler.[41] In all cases gross dilatation of the endolymphatic system, affecting chiefly the scala media (ductus cochlearis B.N.A.) and the saccule and utricle, is present. Minor degenerative changes are found in the organ of Corti and at times in the stria vascularis. In all cases reported, inflammatory changes have been conspicuously absent. This pathologic picture at once disposes of a vast group of disorders that have been termed "pseudo-Ménière's disease," in which the underlying condition is usually inflammation.

Although Lindsay suggested the term "labyrinthine hydrops" for Ménière's disease, he stated that in this type of hydrops the disturbance is confined to the endolymphatic system and the perilymph is not involved. In contrast, the labyrinthine reaction which occurs in serous labyrinthitis affects the perilymphatic spaces primarily while the changes in the endolymphatic system may be limited to dilatation and degenerative changes in the sense organs and stria vascularis.

On the basis of experiments by Hallpike and Cairns, Lindsay felt that the hypothesis of disturbed resorption of endolymph as a cause for idiopathic hydrops appeared unlikely. In his opinion the cause of idiopathic hydrops was more probably a combination of increased production of endolymph and some alteration in its chemical composition, the basis of which he felt remained obscure.

It would seem, therefore, that a more accurately descriptive term for Ménière's disease would be "endolymphatic hydrops." The term "Ménière's disease" because it has been connected with so many different pathologic entities, has become confusing and should be dropped. This is the more appropriate, firstly, because Ménière did not clearly distinguish between the symptoms of endolymphatic hydrops and positional nystagmus with vertigo and, secondly, because he furnished as a probable pathologic basis for the condition the report of a case which did not meet the clinical criteria for the diagnosis which he had established.

The Etiology of Endolymphatic Hydrops. In the course of treating a large number of patients who had endolymphatic hydrops the concept that the cause of this condition is a form of physical allergy was formed. This was owing to the fact that in certain cases of myalgia of the head paroxysmal vertigo was associated with those symptoms and signs considered to be indicative of a causative physical allergy frequently enough so that there appeared to be a definite relationship between the apparent endolymphatic hydrops and the myalgia. I then found that this relationship between myalgia and what appeared to be endolymphatic hydrops had been observed previously by Halle.[42] Since myalgia appeared to be a functional lesion occurring in muscle on the basis of physical allergy, it occurred to me that endolymphatic hydrops might also be a phenomenon attributable to physical allergy. Although many previous observers had suspected this relationship a search of the literature yielded only a little direct evidence in its favor. A few instances have been supplied by Dohlman,[43] Shambaugh,[44] Atkinson[45] and others in which endolymphatic hydrops was demonstrated to be on an allergic basis but in these instances the allergy was definitely of the antigen-antibody or immunologic type. Shambaugh, however, complained of the great difficulty in securing clinical results in the average case by routine methods. Lindsay felt that, while the hypothesis of an allergic basis for endolymphatic hydrops was very attractive, the difficulty in securing any evidence of an immunologic reaction in most cases made it impossible for him to accept the conclusion that the condition was allergic in origin.

Nevertheless the pathologic picture, previously mentioned in reports of histologic studies of endolymphatic hydrops, of a noninflammatory extracellular edema, which the history given by the patient and the recent investigations as to hearing by Crowe, by Mygind and Dederding and by Lillie, Horton and Thornell[46] suggest to be fluctuating in character, is consistent with an allergic basis for this disorder. Indeed it is difficult if not impossible to find a pathologic picture of a fluctuating extracellular edema in the body which does not have an allergic basis.

Acceptance of the hypothesis that there exist two physiologic mechanisms with similar clinical expressions, immunologic allergy and physical or intrinsic allergy, resolves nearly all the difficulties that have prevented endolymphatic hydrops from being generally accepted as a type of allergy.

Duke[47] was the first to postulate the hypothesis of a physical or intrinsic allergy and to suggest that the physiologic mechanism in such cases might differ from the antigen-antibody type of reaction. I have reviewed elsewhere the evidence for considering endolymphatic hydrops to be a type of physical allergy. The gist of the concept that endolymphatic hydrops is a form of physical or intrinsic allergy is that the inherited tendency of the endothelial cells of certain capillary loops to react abnormally to the alarming stimuli of Selye[48] is the anatomic and physiologic basis of the group of conditions known as physical allergy with its disturbance of water and electrolyte metabolism consequent to altered permeability of the cell membranes of the affected cells. This is the capillary picture described by Müller[49] in all types of physical allergy. From review of clinical investigations made of patients who had endolymphatic hydrops it would seem that such alterations were present in this condition.

The hypothesis is presented that the mechanism by which endolymphatic hydrops and physical allergy are precipitated is as follows: A physical or emotional stimulus acting through the parasympathetic nervous system produces an injury to certain cells secondary to the "spastic atonic" change in certain capillary loops which have an inherited tendency to react in this abnormal manner. Owing to the local anoxia produced, cells (probably leukocytes isolated in the capillary loops) are injured, with release of histamine (or the H substance of Lewis). Alteration of the permeability of cell membranes of the capillary wall, transudation of fluid and changes in the metabolism of electrolytes follow.

In reviewing the work of Mygind and Dederding, of Foldes,[50] of Furstenberg, Lashmet and Lathrop,[51] and of Talbott and Brown[52] it has been seen that successful medical treatment of endolymphatic hydrops always has been directed toward correcting the disturbed permeability of cell membranes and disturbed electrolyte metabolism. Clinical investigation again, therefore, substantiates the belief that endolymphatic hydrops is on the basis of physical allergy. I have suggested that physical allergy is a perversion of a normal physiologic reaction, the "alarm reaction" of Selye. It is apparent, however, that physical allergy is not one of the "diseases of adaptation" described by Selye, but rather one of those of "failure of adaptation." Since I[53, 54] have shown that the hormones of the adrenal cortex combined with those of the adrenal medulla can relieve the symptoms of physical allergy, since Kendall[55] has stated that these hormones control the permeability of cell membranes and of water and electrolyte metabolism and since intact adrenal function is necessary for the general adaptation syndrome to develop, I feel that there is good indirect evidence that physical allergy is an alteration of the "alarm reaction," the first portion of this normal physiologic mechanism.

If the evidence from clinical and physiologic investigations and pathologic study is correct, increased fluid pressure originating in the ductus cochlearis secondarily involves the saccule, the utricle and the ampullae of the semicircular canals. Since Dohlman has confirmed Ewald's observation that the horizontal canal reacts maximally when the flow of endolymph in it is in an ampullopetal direction and the flow in the vertical canals is in an ampullofugal direction, the force of the endolymph should act on the cupulas of the canals in either direction, depending on whether conditions are such as to direct the maximal force against the ampullated or the nonampullated openings of the canals. The resulting nystagmus should be rotatory but the direction of the nystagmus woud not indicate the side involved in the process. This is consistent with Simonton's findings in a review of 214 cases of endolymphatic hydrops and his review of the literature as to the direction of vertigo and nystagmus in endolymphatic hydrops.

Histamine Skin Tests. Atkinson, basing his treatment on the original papers of Harris and Moore[56] and of Sheldon and Horton,[57] has used both nicotinic acid and histamine successfully in relieving endolymphatic hydrops. He also adopted the hypothesis of Dzinich and Gallé,[58] who suggested that specific sensitization of an individual to histamine exists and that an intradermal test for sensitivity to histamine would distinguish "histamine-allergic" from "histamine-

nonallergic" individuals. Dzinich and Gallé injected 0.0001 and 0.00001 mg. of histamine in 0.1 cc. of physiologic sodium chloride solution beneath the skin of the forearm. One-tenth cc. of physiologic sodium chloride solution should be injected as a control and the sites of the three injections should be 6 cm. apart. Dzinich and Gallé stated that in allergic individuals the reaction is complete in three to ten minutes. The reaction is positive for allergy when a wheal of 1 cm. and a red areola of 2 cm. in diameter appear at the site of injection.

Atkinson[59, 60] modified the test of Dzinich and Gallé considerably, injecting 0.01 mg. of histamine phosphate contained in 0.05 cc. of physiologic sodium chloride solution. According to Atkinson a positive reaction is the formation of a large wheal measuring 13 to 19 mm. in diameter, surrounded by a wide area of erythema of from 3.8 to 5 cm., and the presence of long, trailing pseudopodia 2.5 cm. or more in length, which appear in three to five minutes, begin to fade only after 20 minutes and are still apparent after 30 minutes. Small buds at the edge of the main wheal which fade in 10 to 20 minutes are not assessed as positive. On the basis of this test, which Atkinson referred to as the test of Dzinich and Gallé, he attempted to divide patients who have endolymphatic hydrops (Ménière's disease) into a histamine-sensitive and a histamine-insensitive group, the one responding to treatment with histamine, the other respondng to treatment with nicotinic acid. Atkinson is inclined to maintain this hypothesis even though all histologic studies on authenticated cases of endolymphatic hydrops show the same pathologic changes. Saucier[61] has attempted to explain away these findings by suggesting that, while in the "vasodilationary" group a primary increase of the endolymph produces both the symptoms and the pathologic picture, in the "angiospastic" group the symptoms are produced by the angiospasm leading to a decrease of the endolymph but the pathologic picture is produced by a "secondary vasodilation" which occurs subsequent to the angiospastic phase.

At the Mayo Clinic 55 patients whose condition was diagnosed as the physical allergy syndrome underwent the Atkinson test and subsequently were treated with either histamine or nicotinic acid without regard to the results shown on the test. The following conclusions were drawn from these tests: (1) There does not seem to be any agreement between the results of skin testing with histamine and the results of treatment. (2) Skin tests with histamine seem valueless both for diagnosis and as a guide to therapy. (3) These findings do not bear out Atkinson's contention that vasospastic and

vasodilating types of allergy exist, since the response of all types in the physical allergy syndrome was equally good to treatment with either histamine or nicotinic acid.

Farmer[62, 63] has studied extensively the role of histamine in allergy. He stated that much evidence points to the conclusion that the symptoms of anaphylactic shock and allergy are due to the liberation of histamine from the tissues of sensitized animals. In his opinion the degree of histamine sensitivity of a species is determined by it adrenal cortex hormone. With regard to Atkinson's hypothesis Farmer stated that with the help of the histamine intradermal test Atkinson feels that one can "satisfactorily separate the histamine-sensitive from the insensitive," the inference being that by this method one can establish whether an individual is allergic or not. On the basis of this reasoning, allergy would consist in increased histamine sensitivity. In Farmer's opinion this assumption is incorrect. He examined for histamine sensitivity a group of normal subjects and a group of persons who had been demonstrated to be allergic by specific skin tests and by clinical examination by means of the test suggested by Atkinson. All tested individuals reacted to the Atkinson test with a distinct wheal which in no instance measured less than 10 to 13 mm. in diameter. To afford Atkinson's conception every possibility of confirmation, Farmer designated as positive a reaction in which there appeared a pseudopod of at least 5 mm. in length. Eighty-six persons were examined, 65 of whom showed positive specific skin reactions for allergy. Only 12 of these latter patients showed a positive histamine reaction according to the foregoing definition. Nine of these 12 patients were suffering from nonallergic diseases; none of them had a positive specific skin reaction; two had positive reactions to histamine according to the Atkinson test. Farmer also came to the conclusion that intradermal histamine tests were of no diagnostic value.

In an extensive review of the literature in regard to histamine, Feinberg[64] stated that attention should be called to the fact that there is a wide range of sensitivity to histamine in different persons and that there is a lack of evidence that the variations are greater among allergic than among nonallergic persons. There is not a sound basis for the belief that allergic manifestations are produced because of a heightened sensitivity to histamine. If histamine has to do with the allergic reaction, it is because an amount above the tolerance dose is released from the tissues. Repeated observations over many years led Feinberg to believe that injections of histamine in man do not increase his tolerance to the substance.

From this review of the literature it would appear that Atkinson's hypothesis that there are two types of persons who have endolymphatic hydrops—the one histamine-sensitive, made worse by nicotinic acid therapy; the other histamine-insensitive, made worse by treatment with histamine—has little if any clinical or laboratory evidence to support it. As I have pointed out, any capillary vasodilator—probably by virtue of its action in opening up the capillary loops involved and sweeping out their contained local excess of histamine, to be diluted and metabolized in the body generally—will produce results comparable to those of any other.

Differential Diagnosis of Endolymphatic Hydrops. Since endolymphatic hydrops is characterized by recurring attacks of either sudden falling or vertigo and nystagmus with loss of hearing and usually tinnitus in the affected ear, differentiation should be confined at least to those conditions which tend to produce recurring attacks of vertigo. One obviously need not consider conditions producing dizziness or unsteadiness, those conditions which produce a continuous vertigo or those conditions in which the patient slowly recovers from a severe initial attack of vertigo and nystagmus without exacerbations. Since the lesion is not inflammatory, recurring attacks of vertigo as observed in cases of chronic suppurative otitis media with fistula into the labyrinth can be excluded at once. Conditions which produce recurring attacks of vertigo on a noninflammatory basis are: (1) trauma of the head with injury to the labyrinth or its tracts; (2) multiple sclerosis (disseminated sclerosis); (3) intermittent tubal occlusion; (4) positional nystagmus; (5) Bruns' syndrome; (6) carotid sinus syndrome; (7) toxic vertigo.

Trauma of the Head.—Grove[65] stated that patients with severe injury of the head and subsequent somatic vertiginous attacks will always be found to have occasional attacks of nystagmus accompanying the attacks. Since vertigo from injury of the head seems to depend on petechial hemorrhages in the region of the vestibular nuclei this vertigo is of the central type and is not to be compared in severity with that occurring in endolymphatic hydrops.

Multiple Sclerosis.—Alpers stated that in multiple sclerosis vertigo is estimated as an initial symptom in 10 per cent of cases. Like other signs of multiple sclerosis it is remittent. Associated with the vertigo and nystagmus is pallor of the temporal sides of the optic disks and absent abdominal reflexes. The vertigo in multiple sclerosis is of the central type and is difficult to distinguish from dizziness on the basis of the history.

Intermittent Tubal Occlusion.—Merica[66] reported 135 cases in which vertigo associated with tubal occlusion was relieved by inflation of the occluded eustachian tube. He stated that the symptoms were usually mild and indefinite but occasionally severe. It seems apparent that Merica made no attempt to distinguish between dizziness and vertigo in the strict sense, so that only the "occasionally severe vertigo" should be considered as being an instance of true vertigo. Merica felt that the patients' tendency to stagger in the direction of the obstructed eustachian tube was a point of diagnostic importance. In my experience patients are seen in whom the symptoms of endolymphatic hydrops (Ménière's disease) are almost perfectly counterfeited by obstruction of the eustachian tube but these cases are rare. In the past five years I have seen only three patients with tubal occlusion in whom it was difficult to make the differential diagnosis from endolymphatic hydrops. These patients were completely and permanently relieved of their symptoms by proper attention to the pathologic condition in the eustachian tube and epipharynx. I believe that all patients who have symptoms suggesting endolymphatic hydrops should have the nasopharynx examined and the patency of the eustachian tubes tested but few will be found to have any abnormal condition in this area. Much of the confusion here is due to inattention to the proper definition of vertigo.

Positional Nystagmus and Vertigo.—Lindsay stated that the term "positional nystagmus" refers to a nystagmus which appears during certain positions of the head or which is greatly influenced by the position of the head. Such nystagmus accompanies the subjective symptom of vertigo occurring in these certain positions which commonly is referred to as "postural vertigo." This symptom with its accompanying nystagmus is known by both animal experimentation and clinical observation to indicate a disturbance in the region of the vestibular sense organs or the central pathways therefrom and is a frequent occurrence in a variety of affections of the central nervous system as well as an occasional accompaniment of a peripheral ear disturbance.

Lindsay felt that a positional nystagmus which is irregular in its direction or which changes direction with changes of position (Type 1 nystagmus of Nylen[67]) appears to be a reliable indication of a centrally located lesion, whereas a positional nystagmus with constant direction (Type 2 of Nylen[67]) may be associated with either a peripheral or a central lesion. Lindsay stated that approximately half the cases of "spontaneous nystagmus" (Type 2 of Nylen) could be ascribed to such factors as hypertension and arterio-

sclerosis, toxic involvement by drugs or infection or some indefinite cause. The other half fell into the group characterized by an abnormally low or labile blood pressure, sometimes with frequent headaches, mild gastro-intestinal disturbances and signs of poor vasomotor control. Lindsay felt that the fact that the vertigo may usually be avoided by changing position slowly seems to support the view that the symptom has as its basis a local circulatory or vasomotor insufficiency. In examining a series of more than 30 cases of the pseudo-Ménière type with positional nystagmus at the time of examination, a Type 1 nystagmus (Nylen) was found in all but one. Lindsay felt that this would appear to be strong evidence of a central location of the disturbance. In examining approximately 66 patients who had endolymphatic hydrops he found only four cases in which positional vertigo could be observed, although on the basis of the history he felt that it was evident that a postural vertigo had existed in a greater number.

Nylen felt that the positions of the head in which it is most important to examine the patient for nystagmus are the right and left lateral positions, the sitting position and the head-hanging position. He stated that nystagmus usually appears in the lateral positions (right or left), in the sagittal positions (sitting and lying) or only in the head-hanging position but sometimes in several of the positions.

Lindsay pointed out that the history of sudden vertigo on change of position is so clear-cut that a diagnosis of positional vertigo can usually be made on that basis alone.

In testing for positional nystagmus I have found that it is sudden angular acceleration about a certain axis that produces the vertigo and nystagmus and not the position per se. In making the test the patient's head should be rapidly changed to the position of test to bring out the nystagmus in most instances. As far as treatment is concerned the group with hypotension and other signs of "chronic nervous exhaustion" respond better to treatment with vasodilators than do patients with endolymphatic hydrops. In the hypertensive-arteriosclerotic group success in relieving symptoms can occasionally be obtained by this method of treatment but failure to relieve symptoms by any method is common. The response to treatment may usually be foretold by examination of the ocular fundi, which usually gives an indication of the degree of arteriosclerosis of the central nervous system.

Bruns' Syndrome.—Bruns[68] originally described episodic attacks of vertigo associated with cysticercosis of the third ventricle. This has now come to include cysts or tumors of the third and lateral ventricles which are characterized by the triad of paroxysms of headache, vomiting and papilledema. True vertigo does not occur in this syndrome but the suddenness of the attacks and the severe nausea and vomiting will produce a confusing picture, since it is often difficult to tell from the history whether vertigo is present or not. Harris[69] found accounts of 49 of these tumors in the literature but felt that these represented only a small proportion of actual cases.

Carotid Sinus Syndrome.—The carotid sinus syndrome is, as pointed out by Weiss, a form of syncope dependent on a rather sudden and usually transient ischemic state of the arterial system. In the classic instance of syncope there is unconsciousness, at times associated with mild clonic convulsions. If, however, the ischemic state of the central nervous system is mild, lightheadedness, weakness, dizziness, scintillating scotomas, dimness of vision, unsteadiness, nausea and vomiting may be the formes frustes. These manifestations are produced by variations of localized pressure within the carotid sinus. Because in instances of hyperactive carotid sinus reflex minor stimuli such as a tight collar may be sufficient to produce the syndrome, the attacks are episodic in nature and are readily confused by the patient with attacks of vertigo.

The carotid sinus test, according to Weiss, must always be performed under identical conditions. The patient should be lying on his back, his head elevated and slightly overhanging a support. If the head is then turned somewhat to one side, the sinus is located just below the angle of the jaw and at a level corresponding to the upper level of the thyroid cartilage. Pressure over this area produces a true depressor nerve reflex. In 60 per cent of normal persons a fall of blood pressure of at least 10 mm. of mercury will result.

Toxic Vertigo.—Toxins from focal infections in teeth, tonsils or sinuses or the toxic effects of certain drugs such as quinine, tobacco, the salicylates and the sulfonamide group may cause episodic attacks of vertigo. Characteristic of these expressions of vertigo, however, is their association with infection or the ingestion of drugs. This type of vertigo may in fact represent a type of hypersensitivity which is obvious when the vertigo is caused by food and in some instances at least may actually represent an attack of endolymphatic hydrops. In any case of recurrent attacks of vertigo a careful search for foci should be made and a careful history of possible exposure to toxic substances should be taken.

In 214 patients reviewed at the Mayo Clinic from 1934 to 1936, inclusive, Simonton found evidences of focal infection of the tonsils, teeth, paranasal sinuses, prostate gland in the male and uterine cervix in the female in one or more foci in 126 (58.8 per cent). This incidence was approximately that found in general admissions and Simonton found it difficult to draw conclusions as to the significance of such infections because of seven patients who had foci of infection removed no report as to results was received from four, two were stated to be improved and one, unimproved. Walsh and Adson[70] found a disappearance of vertigo in 61 per cent of 23 patients who were treated by general measures including removal of foci of infection. They stated that in most of these cases the improvement seemed spontaneous and without reference to any form of treatment. The opinion of Wright that all cases of Ménière's disease depend on focal infection has already been considered. It is impossible to arrive at a definite conclusion in regard to the relation of endolymphatic hydrops to focal infection, but since Lewis showed that sensitization in physical allergy occurs more readily in the presence of infection and since my review of the evidence suggests that endolymphatic hydrops is a form of physical allergy, it is probable that elimination of foci, when present, is important in the treatment of this condition.

Medical Treatment of Endolymphatic Hydrops. Mygind and Dederding reported that, of 157 patients under treatment by their method for two weeks or more, giddiness and nystagmus were relieved in 150 and the hearing was improved in 131. This is a record of success which later methods of treatment have failed to equal. Mygind and Dederding based their treatment on the hypothesis that the symptoms of endolymphatic hydrops are due to increased pressure in the semicircular canals and attributed this increase to a faulty water metabolism which allows water to collect outside the cells, overloading the fluid in the labyrinth. They explained the variability of the symptoms on the basis of a vasomotor phenomenon with parallel increase of capillary permeability. They noted that their patients who had endolymphatic hydrops also suffered from a peripheral disturbance of the circulation as manifested by chilly sensations, cold hands and feet and vasomotor rhinitis, dermatitis factitia, urticaria and the like, phenomena which came and went in periods exactly corresponding to the aural phenomena, particularly the deafness. They noted headache to be a prominent feature in cases of endolymphatic hydrops and described a type occurring in the intervals between attacks in an occasional case which seems similar to the

histamine cephalalgia described by Horton, MacLean and Craig,[71] or in any event a vasodilating type of headache. They felt that endolymphatic hydrops was not a disease sui generis but a typical reaction of a predisposed labyrinth to an almost infinite series of exogenous and endogenous influences which, however, have this in common: that they express themselves through increased capillary permeability.

On the basis of this hypothesis Mygind and Dederding suggested that fluids be restricted to 700 cc. daily, that the ingestion of sodium chloride be greatly restricted so as to promote diuresis and that a reducing diet be given to overweight patients and a high caloric diet be given to underweight patients, and they advised physical therapy consisting of exercise, heat and massage to promote vasomotor tone. They reported that after three years on this regimen 67 of 83 patients had no recurrence of symptoms and all but eight patients had improved hearing. It can be seen that the hypothesis which they present is close to the one which I presented but arrived at by another approach. Mygind and Dederding also anticipated in part the pathologic findings of Hallpike and Cairns.

Successful medical treatment of endolymphatic hydrops has always been based either on the hypothesis that permeability of the capillaries of the stria vascularis was increased or on the hypothesis that fluid or electrolyte metabolism was disturbed. As I pointed out in the section on etiology this is essentially the same as considering endolymphatic hydrops to be a form of physical allergy.

Foldes also based his treatment on the hypothesis that endolymphatic hydrops is caused by a temporary local accumulation of fluids in the labyrinth and felt that it belongs to a group of disorders comprising epilepsy, eclampsia of pregnancy, migraine and allergic diseases. He advocated antiretentional therapy consisting of a high protein, low fat, low carbohydrate and salt-free diet, high vitamin intake, limitation of fluid to 1 to 1.5 liters daily and the administration of 0.25 gm. of quinine sulfate three times a day plus small doses of phenobarbital and nux vomica.

Tobey,[72] as well as Cawthorne and Fawcett,[73] testified to the good results they had with antiretentional therapy.

Furstenberg, Lashmet and Lathrop pointed out that when water accumulates in the body, minerals also accumulate. In a series of controlled experiments they found: that retention of water by the patient would precipitate an "attack" of endolymphatic hydrops; that retention of water without a parallel retention of the sodium ion would not precipitate an attack; that loss of water and retention of

the sodium ion would produce an attack; that loss of water and sodium ion was not associated with an attack of endolymphatic hydrops. From these findings they assumed that it is the retention of the sodium ion and not the retention of water that induces an exacerbation of endolymphatic hydrops. They therefore proposed a low sodium diet with large doses of ammonium chloride to promote diuresis. They reported good results on 125 patients treated in the hospital by means of a sodium-free diet and diuresis produced with ammonium chloride.

Simonton reported that 107 patients with endolymphatic hydrops were treated at the Mayo Clinic with the Furstenberg regimen. These patients were given 14 grains (0.9 gm.) of ammonium chloride three times a day in enteric-coated tablets in alternating courses of three days on and two days off. These patients were not hospitalized for instruction in diet and management but were advised not to add any salt to that used in cooking food and to limit their fluid intake to the equivalent of 15 glasses daily. They were allowed to carry out the treatment at home. Ninety-four patients (87.8 per cent) reported some degree of improvement but only 28 reported complete relief from vertigo while continuing the treatment and 47 patients reported partial relief from vertigo. In other words about 26 per cent obtained worth-while improvement. Among the 28 patients who secured complete relief, the longest interval before recurrence of attacks of vertigo after discontinuing treatment was six months. Unquestionably, better results could have been obtained by close supervision in the hospital but treatment of this sort is impracticable for the average patient.

Talbott and Brown felt that a high intake of potassium may be of more importance in controlling attacks of endolymphatic hydrops than a low intake of sodium. In four cases studied during an attack of endolymphatic hydrops an increased concentration of serum potassium and a decreased concentration of serum sodium were noted. These workers suggested the hypothesis that an increase of serum potassium indicates loss of tissue potassium just as a high level of serum calcium accompanies loss of calcium from the tissues. They felt that such loss of potassium from the tissues would destroy the balance between sodium and potassium in the endolymphatic fluid and that this chemical change might be a factor in precipitating the paroxysms of vertigo characteristic of endolymphatic hydrops. They suggested that the depleted potassium in tissues can be restored and electrolyte equilibrium re-established by administration of additional potassium. They felt that the good results achieved by Furstenberg,

Lashmet and Lathrop[74] depended on the fact that in devising a diet of low sodium content some increase of potassium content is unavoidable. They felt that a diet with normal sodium but proportionally higher in potassium would be therapeutically effective. To achieve this, from 6 to 10 gm. of potassium chloride in an aqueous solution were given daily. Talbott and Brown treated 40 patients with this regimen and reported that all had been relieved but none cured. Most patients experienced an exacerbation of symptoms within a few days after discontinuing treatment with potassium chloride.

Walsh and Adson reported spontaneous remission of symptoms in 5 of 186 patients who had endolymphatic hydrops. They found 36 per cent of 128 patients relieved by a modified Furstenberg regimen, 31 per cent of 13 patients relieved by a low salt diet and potassium nitrate, and 61 per cent of 11 patients relieved by a low salt diet alone.

Harris and Moore suggested the use of nicotinic acid in treatment of endolymphatic hydrops because a large percentage of pellagrins have rotational vertigo. They reported that 17 of 20 patients who had endolymphatic hydrops were completely relieved of symptoms by administration of 250 mg. of nicotinic acid daily in divided doses. In treating 17 patients with endolymphatic hydrops subcutaneously, "good" results were obtained for 16. In treating several hundred patients subsequently by the same technique I have been able to secure complete remission of the symptoms of two thirds of the patients as long as they continued on the treatment (six months). After cessation of treatment, symptoms returned in a few days to six months. An almost parallel result was obtained by treating patients with histamine administered subcutaneously.

After treating 270 patients by intravenous administration of 1 grain (0.065 gm.) of histamine base or its equivalent in from 250 to 500 cc. of physiologic sodium chloride solution, Horton[75] reported complete remission of symptoms in 70 per cent of patients with improvement of hearing in 40 per cent. These patients also experienced a return of symptoms a month to a year after discontinuing treatment.

Schick[76] reported the treatment of 18 patients who had endolymphatic hydrops by the intravenous injection of 5 cc. of a 50% solution of magnesium sulfate. The patients received from two to three injections weekly for a total of 10 to 20 injections. Schick pointed out the danger of intravenous injections of magnesium sulfate and advised that the injections be given at an extremely low rate

with the patient recumbent. Seven of his patients had been free of attacks of vertigo for 14 months.

At the Mayo Clinic we have found that atropine in large doses (up to 1/175 grain [0.00037 gm.]) will have a tendency to control the acute attack of endolymphatic hydrops as will a few minims of 1:1,000 solution of epinephrine given intravenously.

From the review of medical treatment it may be seen that those measures have proved effective which were directed toward correcting the electrolyte balance in the tissue fluids, toward controlling fluid metabolism or toward stimulating the autonomic nervous system and producing vigorous vasodilatation. This clinical evidence all points toward the conclusion that endolymphatic hydrops is an allergic phenomenon, usually of the physical or intrinsic type.

As far as effectiveness of medical treatment is concerned, the matter has never been more concisely stated than by Walsh and Adson, who said: "The medical measures have proved of value and the patient should be given a choice of treatment if he is so situated economically as to afford hospitalization, more or less continuous medical care, and the loss of time if additional attacks of vertigo appear."

Surgical Treatment of Endolymphatic Hydrops. Labyrinthotomy.—In 1871, Knapp[77] compared the causation of endolymphatic hydrops to that of glaucoma of the eye. In 1897, Cheatle[78] expressed a similar opinion and suggested that endolymphatic hydrops may be caused by increased pressure of fluid in the labyrinth resulting either from increased production or from decreased drainage of the perilymph. On the basis of this hypothesis he suggested that endolymphatic hydrops might be relieved by decompression of the labyrinth.

Following this suggestion, Lake[79] in 1906 opened the lateral semicircular canal and vestibule. In 1910[80] he reported complete relief of vertigo in 10 cases of endolymphatic hydrops in which the lateral semicircular canal and the vestibule had been opened on the affected side and in which, in addition, the stapes had been removed.[81]

In 1911 Jenkins[82] made an opening into the lateral semicircular canal with complete relief of vertigo and preservation of hearing. This relief of vertigo continued as long as the fenestra into the labyrinth remained open. Peacock[83] confirmed this observation in 1938.

In 1924 Milligan[84] reported that following labyrinthotomy on the affected side 20 patients who had endolymphatic hydrops were

completely relieved of vertigo, with complete relief of tinnitus in 40 per cent and partial relief of tinnitus in an additional 20 per cent. All patients were completely deaf in the ear on which the operation had been performed.

There were sporadic reports in subsequent years until Day[85, 86] and Cawthorne[87] in 1943 reported a series of cases of labyrinthotomy for endolymphatic hydrops.[88] Day reported complete relief of vertigo in seven cases in which labyrinthotomy followed by cauterization of the membranous labyrinth had been done. Hearing was preserved in a few cases but this hearing has not been retained. In one case in which the origin of the symptoms appeared to be bilateral, partial relief was secured by unilateral labyrinthotomy. Day found that control of the symptoms by medical treatment was readily accomplished after labyrinthotomy.

Cawthorne reported the effects of labyrinthotomy followed by opening the endolymphatic space in 48 cases of endolymphatic hydrops. Relief of vertigo was secured in all of these cases.

In 1946 Day[86] reported the results obtained in fifty additional cases in all of which the patients had secured complete relief. In 1946 Cawthorne reported that uniformly good results had been obtained by this procedure in 400 cases. It is apparent that since the introduction of penicillin labyrinthotomy for endolymphatic hydrops has gained in favor, since there is no longer danger of labyrinthitis following this procedure.

At the Mayo Clinic 21 patients who had endolymphatic hydrops have undergone destructive labyrinthotomy following the technique outlined by Day. In one case the disorder appeared to be bilateral in origin and bilateral labyrinthotomy was done. All the patients who underwent labyrinthotomy were completely relieved of vertigo. One patient on whom unilateral labyrinthotomy had been done reported difficulty in focusing the eyes while at work. The patient on whom the bilateral labyrinthotomy was done has been completely relieved of vertigo but has difficulty with equilibrium when attempting to walk in the dark and a "jumbled" visual panorama when walking or otherwise moving in daylight, although he is free of difficulty when standing still.

Intracranial Division of the Eighth Nerve.—Frazier[89] in 1912 suggested the intracranial division of the eighth cranial nerve for persistent aural vertigo. In 1928 and in subsequent papers Dandy[90, 91, 92] also advocated this method for the treatment of endo-

lymphatic hydrops, which he modified by suggesting hemisection of the nerve, sparing enough fibers of the auditory division so that cochlear function would be preserved.

Crowe, however, reported that in only 22 per cent of the cases in which Dandy had performed hemisection of the eighth cranial nerve (cases which Crowe had subsequently reviewed) was hemisection successful in preserving the hearing in the ear on which the operation had been performed. In the remaining four fifths the hearing either was lost immediately after operation or rapidly deteriorated after the procedure.

One possible reason for this failure to preserve the hearing in the side on which this procedure was performed has been demonstrated by Rasmussen in his study of the funicular pattern of the fibers of the eighth cranial nerve. Rasmussen stated that in only a fair proportion of the nerves that he studied microscopically were two grossly distinct trunks formed. These rarely represented a true division into cochlear and vestibular fibers, for in nearly all such cases there were obvious vestibular fibers in the cochlear trunk. He stated that in one such nerve sectioned nearly a fifth of the apparent cochlear nerve was occupied by vestibular fibers. In his opinion, in resection of what is presumably the vestibular nerve about a fourth of the vestibular fibers would escape being cut. Rasmussen showed that in nerve trunks where no apparent anatomic division is present the division of the fibers is such that many vestibular fibers would remain uncut by the suggested technique. It appears that on the other hand if the surgeon tries to make sure that the vestibular fibers are all divided, not sufficient fibers remain in many cases to carry on cochlear function.

Putnam[93] stated that intracranial section of the eighth nerve is not always as easy, rapid and safe as some accounts would lead one to believe. He advocated a small subtemporal decompression, the dura being elevated as for section of the trigeminal root and the petrosa being drilled anterior to the superor petrosal sinus so as to open the anterior vertical canal. A fine wire is then introduced into the lumen of the canal, which is cauterized with a coagulating current.

Walsh and Adson, in reviewing their results following intracranial division of the eighth nerve, reported that, in those cases in which hemisection was done, when hearing was preserved, vertigo was not abolished and, when vertigo was abolished, hearing was not preserved.

Northington and Barrera[94] studied the responses of monkeys after extirpation of the labyrinth as well as after section of the eighth nerve, unilateral and bilateral. The results were the same whether the labyrinth in question was destroyed or the eighth nerve sectioned.

Although Cairns[95] was one of the first to advocate intracranial division of the eighth cranial nerve in cases of endolymphatic hydrops, he appears to have abandoned this procedure in favor of the labyrinthotomy done by his associate, Cawthorne. From the standpoint of the well-being of the patient it would appear that destructive labyrinthotomy is the procedure of choice. Previous to the introduction of the sulfonamide drugs and penicillin the argument that fenestrating the labyrinth might expose the patient to labyrinthitis by invasion of organisms advancing through the eustachian tube or peritubal lymphatics had some validity, although more than 50 such operations had been reported without such a complication developing. Since the introduction of the antibiotics, however, this fear has no basis as evidenced by the fact that more than 500 labyrinthotomies have been reported without complications due to infection. No death following labyrinthotomy for endolymphatic hydrops has been reported. From the nature of the two procedures labyrinthotomy should be more free from untoward complications than intracranial division of the eighth nerve.

In other words, from the patient's standpoint one has the choice between a major neurosurgical procedure entailing craniotomy and a minor surgical procedure requiring no more than 30 minutes for its performance. I feel that in the United States as in England labyrinthotomy is destined to replace intracranial division. The fact that approximately a fifth of the patients on whom hemisection of the nerve is done subsequently have hearing on the side on which the operation was performed is surprising in view of the fact that the pathologic process appears to have its major expression in the ductus cochlearis and the normal expectation would be continued progress toward complete loss of hearing in the involved ear despite preservation of the cochlear nerve. In selection of patients for surgical tratment, however, those in whom the hearing in the involved ear is still useful and those in whom it can be demonstrated that restoration of hearing to useful levels can be obtained by medical means should not be considered candidates for surgical treatment.

MAYO CLINIC.

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LV

THE ARTIFICIAL MIDDLE EAR

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There are literally thousands of cases of conduction deafness due to chronic otitis media in every large community. Most of these chronic infections resulted from complications following acute exanthemata, which means that the majority of these unfortunate individuals have been deaf since early childhood.

Although the treatment of chronic otitis media has been extensively reported, it is significant to note that few suggestions have been offered for the treatment of the accompanying deafness. Patients with dry middle ears were congratulated on their good fortune and referred to the hearing aid companies for their complaint of deafness. Patients with good hearing and a chronic otitis media that did not respond to local therapy were reluctant to undergo a radical operation except in case of an emergency, for fear their hearing would be further jeopardized by the removal of their diseased but still functioning middle ear. Those that were dissatisfied with the electric hearing aid considered their case hopeless, as they had already run the gamut of treatments with no appreciable improvement in their hearing.

Despite the advances made since the advent of the endaural approach to the middle ear and mastoid, and the Lempert fenestration operation, deafness due to chronic otitis media still remained a problem child of otology.

However, extensive audiometric studies showed that the majority of these patients had excellent bone acuity despite the chronicity of their infection. Actually the deafness was due to an absent or diseased middle ear apparatus which mechanically prevented sound from reaching the cochlea. This led to the idea of replacing the destroyed or nonfunctioning sound conduction apparatus with a unit that would function.

From the Department of Otolaryngology, University of Southern California Medical School.

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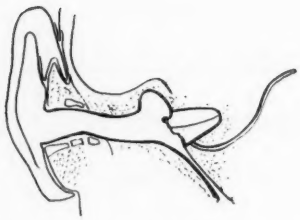

Fig. 1.—Ear drum and outer ossicles missing, with remnants of stapes seen in exposed medial tympani wall.

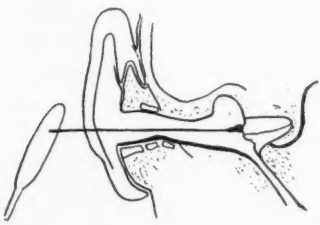

Fig. 2.—Drawing showing medial tympanic wall being explored with acoustic probe.

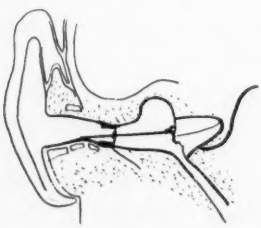

Fig. 3.—Drawing showing artificial middle ear in contact with the oval window.

It is well known that the bird has extraordinary hearing. Examination of the bird's middle ear reveals that it is quite similar to that of the mammal except that the ossicular chain is represented by a single sticklike structure known as the columella.[1] Since it would be extremely difficult to construct and introduce a middle ear apparatus patterned after the mammal, the possibility of replacing the lost sound conduction system in the human being with a unit patterned after the bird's ear presented itself.[2]

A crude bird's middle ear was made in the form of an acoustic probe. This consisted of a diaphragm of x-ray film which supported a slender stem of bamboo, tipped with a small ball. The stem of the acoustic probe was introduced into the ear canal through a speculum and the exposed medial tympanic wall was explored by touching it lightly here and there while the examiner kept talking. Theoretically, if a yielding area related to the vestibular space was contacted, the patient should immediately declare that the examiner's voice had suddenly become loud.[3] In other words, the acoustic probe had temporarily replaced the nonfunctioning or absent human middle ear, and the loudness of the examiner's voice was due to the vibrations of the diaphragm being transmitted down the stem to the yielding area of the medial tympanic wall.

The results with the acoustic probe in cases of chronic otitis media were very dramatic. The patient was able to hear a conversational voice in an adjoining room with the probe in place. A positive reaction was always elicited when contact was made with a yielding area providing the patient had adequate bone acuity.

Quite obviously the acoustic probe was not practical except for its use as a diagnostic aid and to determine accurately the location of the yielding area. If the patient responded positively to the acoustic probe, he was theoretically a candidate for a smaller unit patterned after the bird's middle ear.

A simple imitation of the bird's sound conduction apparatus was constructed by using a small, plastic, cylindrical insert which had been accurately fitted to the external bony canal. A fish skin was stretched over the cylinder by means of a detachable silver ring to make a tense diaphragm, and a flexible rod of nylon tipped with dental stent was fed through a reinforced hole in the diaphragm to imitate the columella of the bird. The cylinder was then inserted into the external bony canal and the rod was introduced into the middle ear until the tip came into contract with the yielding area.

Fig. 4.—Enlarged artificial middle ear showing detachable silver ring, drum membrane and tipped nylon rod.

When proper contact was made, the results were as dramatic as those obtained with the acoustic probe[3] (Figs. 1-4).

Cases 1, 2, and 3 had bilateral chronic otitis media since early childhood. There was a loss of ear drum and ossicles on both sides with dry tympanic cavities. Their bone acuity despite the chronicity of the infection was excellent and all responded positively to the acoustic probe. Audiograms following the insertion of the prosthesis revealed that it had little effect at the low frequencies but produced a dramatic boost at the higher frequencies, especially at 1024 and 2048 c.p.s. The cases presented showed an average improvement of 10 decibels at 128 and 256, 20 decibels at 512, and from 30-35 decibels at 1024 and 2048. Seemingly the prosthesis does not transmit low frequency adventitious noises and confers the greatest improvement in the consonant range, resulting in a disproportionate increase in the intelligibility for the spoken voice (Figs. 5-7).

However, when more patients with chronic otitis media were examined with the acoustic probe, it was noticed that there were usually two contact areas which elicited positive reactions in an exposed middle ear. These two areas corresponded to the oval window and the round window niche. Some patients responded only at the oval window, while others responded only at the round window niche. Those who responded at both windows remarked on the difference in quality of the sound when examined with the acoustic probe. The examiner's voice seemed lower and more resonant when the round window niche was contacted.

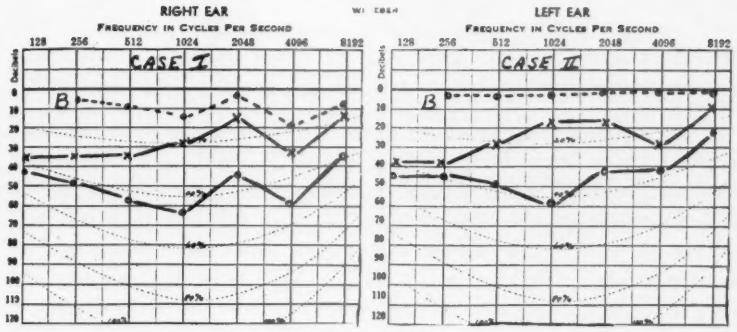

Fig. 5.—Audiograms of Case 1. Note that the insert has little effect at the low frequencies, and the dramatic boost at the higher frequencies, especially at 1024 and 2048.

B . - - - . Bone acuity curve
x———x Air acuity curve with artificial middle ear
o———o Air acuity curve without insert

Fig. 6.—Audiograms of Case 2.

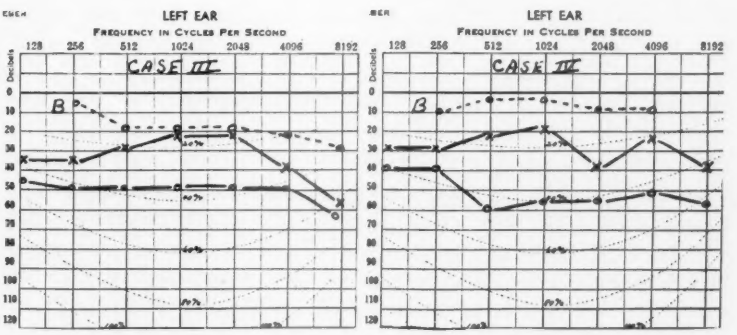

Fig. 7.—Audiograms of Case 3.

Fig. 8.—Audiograms of Case 4. The middle ear prosthesis has been fitted to the round window niche with the maximum response at the 512 and 1024 levels.

By separating those who had only one contact area it was noticeable that the audiograms of those with prostheses fitted to the oval window followed the pattern described above, in that there was little effect on the sensitivity of lower frequencies with the maximum boost at the higher frequencies, mainly 1024 and 2048.

Those fitted with inserts to the round window showed a different type of audiometric curve. Cases 4 and 5 reveal an improvement throughout the frequency range with the maximum response at the lower frequencies; the improvement averaged from 30 to 35 decibels at 256 and 512. It is interesting to note that Case 5 was fitted on both sides with a middle ear prosthesis and that the oval window was contacted on the left side and the round window niche on the right; the different type of response obtained in each ear seems to be characteristic of each window (Figs. 8-10).

Case 6 had a history of chronic recurrent otitis media in early childhood. A right radical mastoidectomy was performed approximately 25 years ago. Since then the right ear has been dry. His hearing has been poor since the operation and the patient states that he hears only with his left ear which has drained intermittently for the past 10 or 15 years. Examination revealed a well healed excavated right radical mastoid cavity. The left ear presents a chronic purulent otitis media with a perforated scarred ear drum. The left ear was only slightly positive to the acoustic probe and the right ear did not react at all. However, closer examination of the radical cavity revealed that a thick false membrane was acting as an insulating curtain and that it also concealed the contact area from the probe. The patient consented to a surgical removal of the membrane which was done under local anesthesia. He experienced an immediate improvement in hearing as soon as the scar tissue was incised. When the false membrane was removed, he responded very positively to the acoustic probe. At this time it was determined that his contact area was the round window. He was fitted with an insert with very satisfactory results and the typical curve with the maximum boost at the lower frequencies was obtained (Fig. 11).

The proper fitting of the diaphragm rod prosthesis is not as simple as it seems. It is necessary to have as near perfect a cast of the ear canal as possible so that no irritation will develop from an improperly fitting insert. It is also important to take into consideration the motility of the ear canal caused by condylar displacement. This is accomplished by having the patient chew gum while the impression of the ear canal is being taken. The rod must be

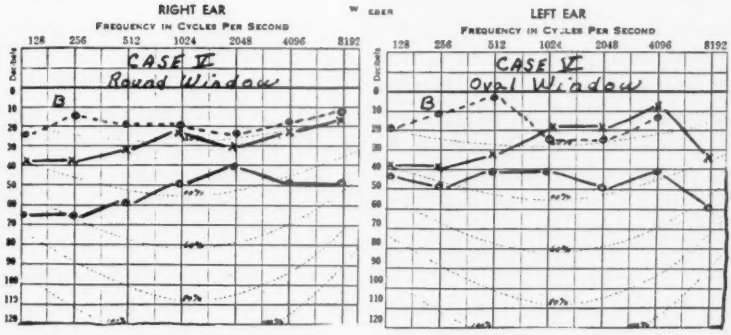

Fig. 9.—Audiograms of Case 5. Case of chronic bilateral otitis media with loss of ear drum and ossicles. The left ear was fitted with an insert to the oval window, and the right ear was fitted with an insert to the round window niche.

Fig. 10.—Audiograms of Case 5. Case of chronic bilateral otitis media with loss of ear drum and ossicles. The left ear was fitted with an insert to the oval window and the right ear was fitted with an insert to the round window niche.

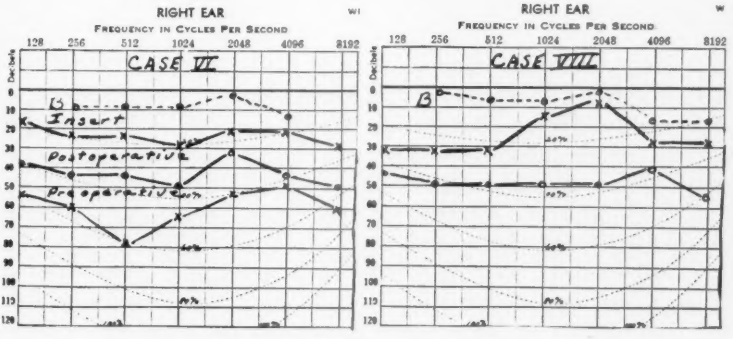

Fig. 11.—Audiograms of Case 6. There was a spectacular response to an insert fitted to the round window niche in a radical mastoid cavity following a plastic operation to remove a false membrane that concealed the medial tympanic wall from the acoustic probe.

Fig. 12.—Audiograms of Case 8, showing the effect of a middle ear prosthesis on a fenestration case in which there was no appreciable improvement in hearing following surgery.

sufficiently short to make proper contact and not to project too far beyond the outer limit of the insert as it might dampen itself on the external ear canal. These requirements make it difficult to apply the prosthesis in contracted or narrow canals, especially those in which the cartilage of the helix overhangs the external orifice. It is also extremely difficult to fit a prosthesis in cases in which the external canal has been widely excavated through removal of too much of the posterior canal wall, and in which the external orifice is considerably smaller than the cavity into which the prosthesis must be placed.[3]

The insert has been worn as long as four to six weeks without any sign of irritation, but the accumulation of secretion tends to load the diaphragm and decrease the effectiveness of the device.[3] For this reason the patient is taught to introduce, remove and repair the insert, so that he is completely independent of the doctor in case of a mishap. Therefore, many eligible candidates for the prosthesis require some type of plastic operation to facilitate the introduction of the middle ear insert.

Those requiring a radical operation no longer have to fear a further loss of hearing but can be assured an improvement over their preoperative acuity level, providing a proper contact area for the prosthesis has been established. When the removal of the middle ear apparatus is indicated, the bony and cartilaginous canal should be left in such a condition that proper placement of the prosthesis may be effected by the patient.

Through the cooperation of other otologists patients who had had fenestration operations and had not obtained a permanent improvement in hearing were examined with the acoustic probe to determine the possibility of fitting them with a diaphragm rod prosthesis to the area of the artificial window. The majority of these patients obtained no response to the acoustic probe, substantiating the viewpoint that osteogenesis had closed the window. However, extremely positive reactions were obtained in three cases demonstrating that the fistula was probably patent, but either because of some interference with the flap or scarring of the membranous labyrinth these patients were unable to obtain useful hearing.

Case 8 obtained no improvement in hearing following the fenestration operation. He also suffered severe vertigo for four months and the ear drained for approximately twelve months following the operation. He still complains occasionally of vertigo, especially following sudden head movements. Examination revealed

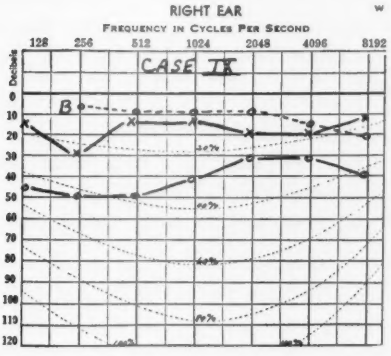

Fig. 13.—Audiograms of Case 9. Air acuity curve following contact to the artificial fistula with a middle ear insert.

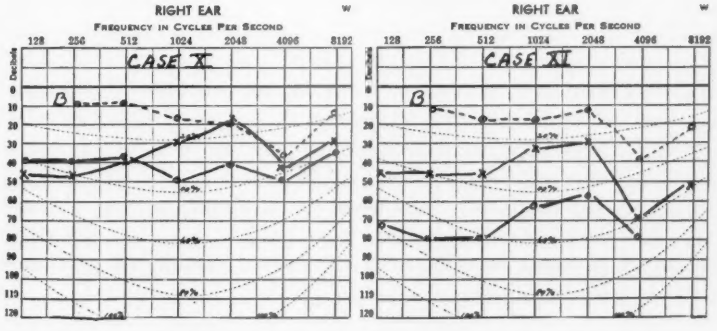

Fig. 14.—Audiograms of Case 10. Air acuity curve following contact to the artificial fistula with a middle ear insert.

Fig. 15.—Audiograms of Case 11.

a well-healed Lempert cavity. There was a dramatic improvement in hearing when the fistula was contacted by the acoustic probe. It was interesting to note that this contact area was pinpoint in size and the slightest deviation from this region with the acoustic probe elicited no reaction.

Cases 9 and 10 obtained useful hearing for three to four months following the window operation and then their hearing gradually dropped back to its preoperative level. Both of these patients had a positive fistula test and reacted very favorably to the acoustic probe.

These three were fitted with a diaphragm rod prosthesis. The audiograms show that the hearing of Cases 9 and 10 compares favorably with that of the optimum postoperative cases. Case 10 has reverted back to his hearing aid as he feels the insert does not give him sufficient hearing. However, this may be explained by his inadequate bone conduction curve (Figs. 12-14).

Case 11 had a bilateral fenestration operation with no improvement in hearing. Examination revealed on the left side a well-healed Lempert cavity which was negative to the acoustic probe. However, on the right side the ear drum had sloughed away, exposing a clean medial tympanic wall with the stapes and round window niche in full view. The stapes area showed no response to the probe, substantiating the diagnosis of otosclerosis. Contact over the artificial window caused severe vertigo with no improvement in hearing. However, a very positive reaction was obtained when the round window niche was explored. The round window was fitted and a typical response was obtained in that there was a pronounced boost throughout the frequency range with the maximum gain at 256 and 512. Unfortunately, the patient was unable to wear the prosthesis because of the extreme vertigo caused by the pressure of the insert in the region of the artificial window (Fig. 15).

SUMMARY

The acoustic probe should be employed on all patients whose drum membrane and ossicles are absent or have been removed to determine whether a contact area for the prosthesis is available.[3]

An ossiculectomy or a radical operation should be considered for all patients with perforated drum membranes and discharging ears whose condition does not yield to treatment, providing a proper contact area for the prosthesis has been established. When the re-

moval of the middle ear apparatus is indicated, the bony and cartilaginous canal should be left in such a condition that proper placement of the prosthesis may be effected by the patient.[3]

Patients who have undergone the Lempert fenestration operation with no improvement in hearing or who have obtained an improvement that is below the useful serviceable level should be examined with the acoustic probe to determine whether they could be candidates for the diaphragm-rod prosthesis.

The diaphragm-rod insert is invisible and its efficiency even exceeds that of a vacuum tube hearing aid in favorable cases.[3] If it is fitted to the oval window, it has little effect on the low tones and may increase sensitivity at the higher frequencies by as much as 40 decibels. If fitted to the round window, there is improvement throughout the frequency range with the maximum improvement averaging 30 or 35 decibels at 512.

The simplicity of the prosthesis should appeal to all otolaryngologists, as no intensive training is required to master the technique of its use.

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LVI

TOLERANCE FOR PURE TONES AND SPEECH IN NORMAL AND DEFECTIVE HEARING

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The traditional approach to measurement of impairment of auditory function has, in the main, involved measurements of threshold acuity for either pure tones or speech. The former approach has particularly characterized clinical practice and procedures. It has been demonstrated, however, through the study of loudness well above threshold that the threshold audiogram alone is not an adequate measure of the impairment of auditory function.[1] Furthermore, the impairment of understanding of speech is more closely related to the over-all loudness loss at the intensity level at which speech is heard than to the threshold audiogram.[1] It is, therefore, difficult to predict from a threshold-of-acuity measure how the impaired ear will function in an above-threshold acoustic environment.

The need for accurate information concerning responses well above the threshold of acuity in order to appraise adequately auditory function has consequently focused attention upon the concept of the *auditory area* or auditory "map." The auditory area is properly described by equal-loudness contours[2] and is bounded at its lower border by the threshold of acuity and at its upper border by the thresholds of tolerance. The auditory area might be likened to a building with the foundation represented by the thresholds of acuity, the in-between stories corresponding to levels of equal loudness, and the roof represented by tolerance limits.

The investigation described in this report represents a systematic study of the quantitative and qualitative nature of the tolerance limits

This paper is an abstract of O.S.R.D. Report No. 6303, 31 July, 1946, completed under a contract between the Office of Scientific Research and Development and the Central Institute for the Deaf. The original report is entitled "Tolerance for Pure Tones and Speech in Normal and Hard-of-Hearing Ears" by S. Richard Silverman, C. E. Harrison and H. S. Lane. The abstract constituted a paper delivered to the American Laryngological, Rhinological and Otological Society at its meeting in St. Louis, Missouri, April 25, 1947.

The writer also wishes to acknowledge his indebtedness to Dr. Hallowell Davis, who represented the Office of National Defense Research Committee as supervisor of the project.

for speech and pure tones of normal and deaf human ears. With reference to the Aural Rehabilitation Programs of the Armed Forces, it was hoped to attain the following significant objectives:

1. To yield data pertinent to the design characteristics of future hearing aids. The maximum acoustic output of present-day hearing aids, either by intent or accident, is such that presumably it protects the wearer from acoustic stimuli that would exceed his tolerance limits. If it could be demonstrated that the tolerance limits are higher than heretofore supposed, the instrument could be designed with a higher level of maximum undistorted acoustic output and thereby the auditory range of usefulness could be increased materially.

2. To provide information which could guide procedures in the clinical selection of hearing aids.

Previous studies[3] indicate a relatively vague conception of the qualitative nature of the threshold of pure tone tolerance. This threshold has at times been termed the threshold of "pain," and more frequently has been called the threshold of "feeling." From the psychophysical standpoint the "pain" response is most readily identifiable but the "feeling" response is vague. The subject is at a loss to know when "hearing" ceases and "feeling" begins. Furthermore, these last two sensations can, and do, occur simultaneously as the clinician who practices bone conduction audiometry can testify.

In the experimental approach to the determination of pure tone tolerance, the present investigators, therefore, postulated several qualitatively different tolerance thresholds which could be established in terms of separately identifiable sensations. It was felt, in other words, if the analogy of the auditory area and the building is carried out, that the "roof" of the auditory area might be multilayered in character. Looking upwards the roof of the building might be comprised of a plaster ceiling, supporting beams and an external roof. It was supposed that the responses of the subject to high intensity stimuli might reflect a pattern of multiple layers varying with the qualitative nature of the response to be elicited.

Exploratory experimentation suggested that three specific pure tone tolerance threshold layers could be established as a function of frequency and intensity. It was determined further that the same thresholds could be used to establish specific levels for speech tolerance.

Three clearly distinguishable thresholds proved to be:

1. *Discomfort threshold,* defined as the point at which the subject feels that he would cease to care to listen because the stimulus was uncomfortable. (The precise instructions to the subject are given later in the report.)

2. *Tickle threshold,* defined as the point at which the subject experiences a definite tickling sensation in the ear.

3. *Pain threshold,* defined as the point at which the subject experiences a definite sensation of sharp pain, as opposed to more discomfort deep in the ear.

ORGANIZATION OF EXPERIMENTS

The experiments were organized in the following general manner: (Details of procedure are given later.)

1. *Pure Tone Tolerance*—The subjects were divided into two groups, normal hearing* and hard-of-hearing. There were 16 ears in each group exposed to the complete experimental procedure. At each experimental session the thresholds on each ear for discomfort, tickle, and pain were determined for given frequencies and then repeated. In other words the thresholds were determined for the right ear, then the left ear, followed by a repeat series of measurements on the right ear and the left ear. Except in a few instances there were six consecutive sessions one week apart for each subject. In most cases the threshold of acuity was determined before and after testing for tolerance thresholds. This latter procedure was introduced in the course of the experiment to determine what effect exposure to high intensity stimuli might have on the threshold of acuity.

2. *Speech Tolerance.*—There were 30 normal ears and 30 hard-of-hearing ears exposed to the complete experimental procedure. These subjects were subdivided into three groups (1, 2, and 3) of 10 normal and 10 hard-of-hearing ears each. The basis of division between Groups 1 and 2 was the order of testing of ears with the time interval between sessions remaining constant. Groups 1 and 3 differed in the time interval between sessions with the order of testing of ears remaining constant. An experimental session consisted of determination of thresholds of acuity for speech, and discomfort, tickle, and pain thresholds four times for each ear, All subjects were exposed to four sessions of speech tolerance testing as contrasted with six sessions for pure tone tolerance. The fact that the speech

*The "normal hearing" group will subsequently be referred to as "normals."

tolerance threshold curves were reaching asymptotes as a function of experience determined the number of sessions.

3. *Study of Change of Tolerance.*

a) Change with Experience: The change of tolerance (which subsequently proved to be upward) for pure tones and speech as a function of the number of consecutive testing sessions was studied.

b) Retention of Tolerance: The retention of tolerance for speech as a function of given time intervals from the date of the last test was investigated.

c) Contralateral Tolerance: The effect of tolerance tests for speech and pure tones of one ear on the opposite ear was studied. This phenomenon is defined as contralateral tolerance.

d) Transfer Tolerance: The effect on tolerance for pure tones as a result of exposure to speech tolerance procedure was investigated. This phenomenon is defined as transfer tolerance.

e) Methods of Elevating Tolerance: When it was noted that the testing procedure, per se, elevated tolerance, it was decided to make a deliberate experimental attempt to increase tolerance in hard-of-hearing ears through exposure of the subject to two different levels of high intensity speech. Therefore, a third group of 10 hard-of-hearing subjects was exposed at four weekly experimental sessions. The right ears of this group were exposed to one level of stimulation and the left ear to another level.

SUBJECTS

Equal numbers of normal and hard-of-hearing subjects were chosen for the experiments on pure tone and speech tolerance. There were 16 ears in each group for pure tone tolerance and 30 ears in each group for speech tolerance. In the study of experimental elevation of tolerance, 20 hard-of-hearing ears were involved. Thus, a grand total of 46 normal ears and 66 hard-of-hearing ears were employed throughout the entire series of experiments. These figures do not include a few subjects (noted later) who dropped out, for one reason or another, during the course of experimentation.

The classification of subjects into the following clinical types of deafness was established after thorough otological examination. The criteria are also given:

1. High frequency nerve deafness—abrupt loss in hearing for air and bone conduction, beginning at 1000 cycles.

2. Nerve deafness—marked reduction in hearing for air and bone conduction.

3. Conduction deafness—normal hearing or slight decrease in hearing for bone conduction with greater loss for air conduction. (These subjects usually show a negative Rinne test and a normal or retracted tympanic membrane. The pathologic condition involved is usually early otosclerosis or hyperplastic otitis media.)

4. Mixed Deafness—bone conduction slightly better than or equal to air conduction. (The pathologic condition is usually a combination of early nerve lesion and middle ear disease.)

The hard-of-hearing subjects represent a fairly well balanced distribution of clinical types. Detailed information concerning the subjects in each group is given in appropriate sections of the complete report.

APPARATUS

The apparatus is described in the complete report.

PURE TONE TOLERANCE

Instructions to Subjects. The subject was seated in a comfortable chair situated within the sound chamber. Instructions were communicated directly except in cases of severe deafness, when the instruction microphone was used to insure accurate understanding of directions. The precise instructions for the three thresholds were as follows:

1. *Discomfort*: "You will hear a tone which will get louder and louder. Tell me when you reach the point where the tone is uncomfortable, that is, when you would no longer care to listen or when you feel like removing the earphone from your ear. When the uncomfortable point is reached, say 'uncomfortable' and I will shut off the tone. We shall then repeat the procedure with another tone. Are you ready?"

2. *Tickle*: "You will hear a tone which will get louder and louder. Tell me when you reach the point where you feel a tickling sensation deep in the ear as though a broom straw were tickling it. Be alert only for the tickling sensation. When the tickle point is reached, say 'tickle' and I will shut off the tone. We shall then repeat the procedure with another tone. Are you ready?"

3. *Pain*: "You will hear a tone which will get louder and louder. Tell me when you reach the point where you feel a sharp pain deep in the ear. Be alert only for the pain sensation. When the pain point is reached, say 'pain' and I will shut off the tone. Are you ready?"

In cases where the subject failed to comprehend the meaning of the instructions, they were repeated or elaborated upon with the basic point in mind that the type of response (for discomfort, tickle and pain) desired was not altered. Obviously, as the subject became more sophisticated, the necessity for repetition and elaboration of instructions decreased.

Frequencies Tested. The following frequencies were tested: 250, 500, 1000, 1400, 2000, 2800, 4000 and 5600 cycles per second. These frequencies were chosen because they involve an adequate sample of the speech range and because they include the frequency range of all hearing aids. Also these frequencies were best suited to the frequency response of the PDR-10 earphone which has a fairly flat response throughout the spectrum chosen for testing. Exploratory tests showed that because of the downward slope of the frequency response of the PDR-10 earphone beyond 5600 c.p.s., tolerance limits could not very often be reached in that area. Furthermore, below 250 c.p.s. the seal of the earphone against the ear is critical. A slight leak may introduce a large error.

Duration of Exposures and Stepwise Increases of Intensity. The subject was exposed at each frequency, starting at a level of 100 db. above 0.0002 dynes/cm.^{2} Each exposure lasted for 1.5 seconds and then the intensity was increased in 2-db. steps without interrupting the tone. The step intervals of 2 db. were maintained up to 130 db. If the subject had not yet reached his tolerance, the step intervals were reduced to 1 db. The duration of each step was maintained at 1.5 seconds. The tolerance limits of some subjects exceeded the limitations of the apparatus (approximately 145 db. r.m.s. at 1000 c.p.s.). The technique of determining tolerance limits by a method of gradually increasing intensities appeared to be appropriate since we wished to avoid the complications introduced by the sudden onset of a high intensity stimulus. A level of 100 db. above 0.0002 dynes/cm.^{2} was adopted as a convenient starting point. Only a few subjects (on early tests) reported discomfort below 100 db.

Sequence of Observations. The following sequence of frequencies was used: 1000, 1400, 2000, 2800, 4000, 5600, 500, 250 c.p.s. The sequence is arbitrary but follows orthodox audiometric

technique in commencing with the portion of the frequency spectrum to which the ear is most sensitive. The discomfort thresholds were first determined at the various frequencies, then the tickle thresholds, and finally the pain thresholds.

Thresholds of Acuity. Beginning about midway in the experiment the thresholds of acuity at each frequency were determined before and after the tolerance tests. In other words, the thresholds of acuity from 250 to 5600 c.p.s. were obtained (using the same apparatus), then the threshold for discomfort, tickle and pain, followed immediately by another determination of the thresholds of acuity.

Order of Ears Tested. The right ear was always tested first, followed immediately by the left ear.

Testing Timetables. Each testing session consisted of a complete series of tolerance tests on each ear (Test A), followed after a ten-minute rest period by a repetition of the complete series on each ear (Test B).* An experimental session lasted from 105 to 120 minutes. These sessions were repeated at weekly intervals for six weeks. This schedule allowed a comparison of the effects of brief as well as the much longer (one week) intervals between tests.

Subjects. There were 9 normal and 10 hard-of-hearing subjects in the pure tone tolerance experiment. However, the 9 normal subjects represented only 16 ears since only one ear of each of two subjects was used. Midway in the experiment two hard-of-hearing subjects dropped out, leaving a total of 8 subjects (16 ears) who completed the experiment.

RESULTS—NORMALS

Shift of Tolerance Indices with Experience. It is evident from Fig. 1 that the three thresholds are systematically elevated as a function of number of sessions. The amount of elevation for the discomfort threshold was 10.1 db. from Session IA to VI. However, it is impossible to know the amount of elevation of the tickle and pain thresholds because the limits of the apparatus were reached prior to Session VI. The elevation of the tickle threshold from Session IA to IVA was 8.8 db. and for the pain threshold it was 1.4 db. from Test IA to IB. It is obvious, therefore, that the amount of elevation for tickle and pain is greater than these values.

*A few subjects had only A tests at the outset of the experiment. In a few cases, the weekly sequence was interrupted for approximately three months after the third session before experimentation was resumed. These irregularities are indicated in the data.

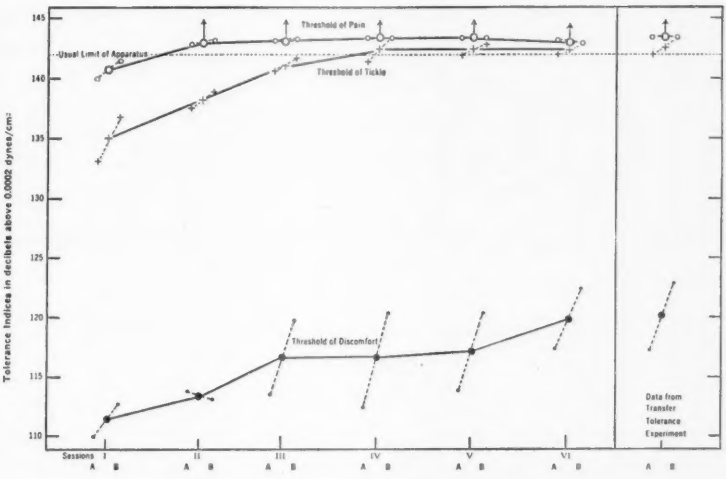

Fig. 1.—Elevation of tolerance indices for pure tones—Normals. Inter-session shift indicated by solid line; intra-session shift by dotted line.

Auditory Map. The ultimate threshold levels of tolerance for discomfort, tickle and pain are represented by the asymptotes of the three curves in Fig. 1, showing the means of each session. By fitting the shapes of the curves previously derived to the nearest asymptotic values, the final tolerance thresholds can now be represented by the curves in Fig. 2.

It should be recalled that the discomfort level is represented by the mean of a curve having a long shallow minimum from 1400 to 4000 c.p.s. which is approximately 3 db. below the mean for all frequencies. Using the mean points for each session to determine the asymptote the mean value for the ultimate discomfort level is 120 db.

From Fig. 1 it is clear that the ultimate tickle threshold cannot be established precisely because the approximate mean value for later tests lies beyond the limits of the apparatus and, therefore, the asymptote is indefinite. All that can be said is that the tickle threshold is greater than 141.9 db., which represents the last approximate mean lying below the range of the apparatus.

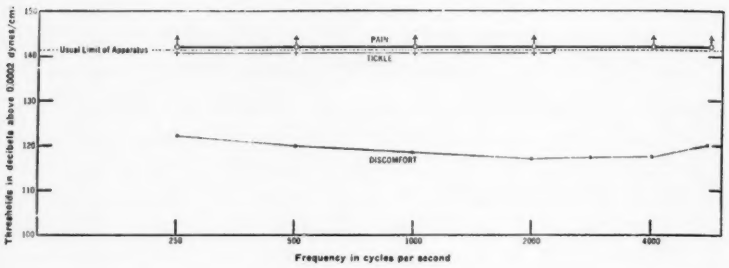

Fig. 2.—Ultimate thresholds for pure tones—Normals

The asymptote of the pain threshold in Fig. 1 is undoubtedly an artefact since the approximate mean as early as Session IIA lies beyond the apparatus limitations. It is certain, however, that the mean threshold is greater than 141.5 db.

Fig. 3 represents the auditory map using the data of Sivian and White[4] for the threshold of acuity. For purposes of comparison Wegel's[3] points for the thresholds of "feeling" and an equal loudness contour at the 100 db. level[2] are included. The uppermost curve labelled "threshold of pain" actually indicates the limit of the apparatus, and the vertical arrows indicate that the actual threshold of pain lies somewhere slightly above this line. The map suggests the following comments:

1. The final levels, never before approached because of inadequacy of equipment, are surprisingly high.

2. The shape of the discomfort contour is supported by the data of Wegel,[3] Lierle and Reger,[5] and others. The levels suggest that Wegel's threshold was the beginning of the tickle sensation. This inference is supported by Wegel's description of his instructions to subjects.

3. The shape of the discomfort contour strongly suggests that it is a high-intensity equal-loudness contour.

4. There is no support for the theory that high-pitched tones are more painful, as has been suggested by Fletcher's curve.[4] Al-

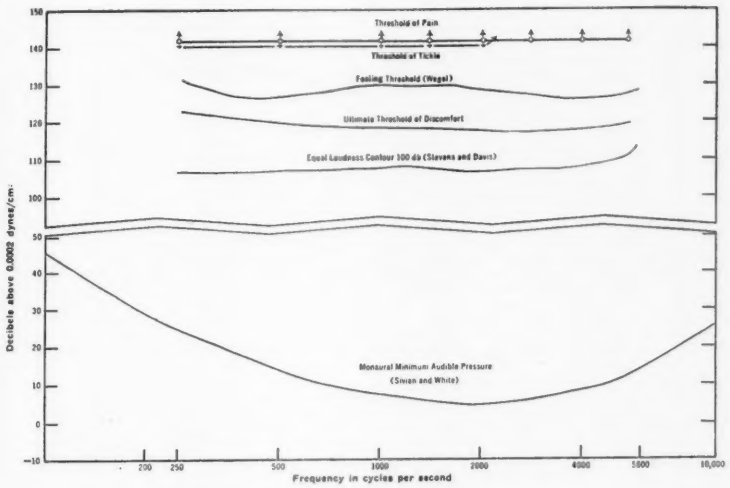

Fig. 3.—Auditory map for pure tones—Normals. The curves in the upper half of the figure represent pressures measured under the receiver by means of a coupler. The lower curve for minimum audible pressure has been corrected to give the pressure at the ear drum. It is therefore approximately but not precisely comparable with the upper curves.

though because of limitations of apparatus it was impossible to obtain data for the area around 8000 c.p.s., there is no suggestion of a downward trend in the higher frequencies. The reports that higher frequencies are more painful are probably the result of semantic confusion with such sensations as "sharp," "piercing," and "annoying."

RESULTS—HARD-OF-HEARING

Shift of Tolerance Indices with Experience. Using the single index of tolerance (mean of medians for all frequencies), the systematic elevation of threshold as a function of the number of sessions is again shown in Fig. 4. The dip at Session IV reflects a time break of approximately three months for all but one subject. The ultimate elevation of the discomfort threshold was 10.7 db. The elevation for the tickle threshold from Session I to IVB was 11.7 and for pain from Session I to IIA, 5 db.; the ultimate elevations must have been still greater.

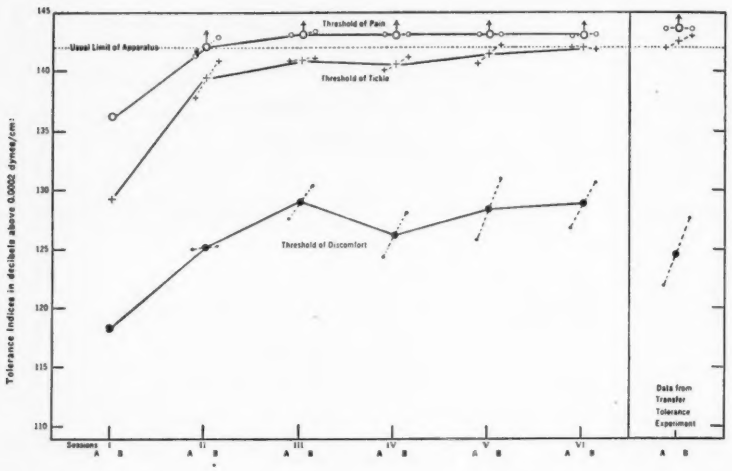

Fig. 4.—Elevation of tolerance indices for pure tones—Hard-of-Hearing. Inter-session shift indicated by solid line; intra-session shift by dotted line.

Auditory Map. The ultimate threshold levels of tolerance for discomfort, tickle, and pain are the asymptotes of the three curves (showing the means of each session) in Fig. 4. By locating the shapes of curves previously derived at the appropriate mean levels, the final tolerance thresholds are represented by the curves in Fig. 5. The ultimate discomfort level is 129.1 db. The ultimate tickle threshold cannot be established precisely because the approximate mean value for later tests lies beyond the limits of the apparatus; therefore, the asymptote is indefinite. All that can be said is that the tickle threshold is greater than 141.1 db. which represents the last approximate mean lying below the range of the apparatus. The pain threshold is greater than 141.3 db.

The high level of the tickle and pain thresholds suggests that there is an approachable and potentially useful portion of the auditory area beyond the range of present audiometry. Consequently, some individuals who have heretofore been termed "totally deaf" as a result of audiometric tests might be reached by auditory stimulation through a properly designed apparatus.

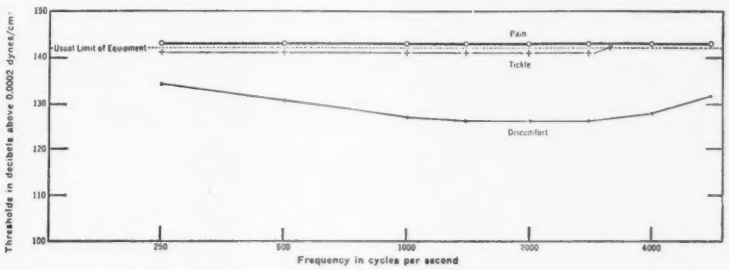

Fig. 5.—Ultimate thresholds for pure tones—Hard-of-Hearing.

SPEECH TOLERANCE

Instructions to Subjects. The same precautions, as in the previous experiment, were taken to insure accurate understanding of instructions. The precise instructions for the three thresholds were as follows:

1. *Discomfort*: "You will hear a man talking and his speech will get louder and louder. Tell me when you reach the point where the speech is uncomfortable; that is, when you no longer care to listen or when you feel like removing the earphone from your ear. When the uncomfortable point is reached, say 'uncomfortable' and I will shut off the speech. You are not required to remember the content of the talk. Are you ready?"

2. *Tickle*: "You will hear a man talking and his speech will get louder and louder. Tell me when you reach the point where you feel a tickling sensation deep in the ear as though a broom straw were tickling it. Be alert only for the tickling sensation. When the tickle point is reached, say 'tickle' and I will shut off the speech. You are not required to remember the content of the talk. Are you ready?"

3. *Pain*: "You will hear a man talking and his speech will get louder and louder. Tell me when you reach the point where you feel a sharp pain deep in the ear. Be alert only for the pain sensation. When the pain point is reached, say 'pain' and I will shut off the speech. You are not required to remember the content of the talk. Are you ready?"

Speech Material. The speech material consisted of a recorded sample of connected discourse from a news broadcast by Fulton Lewis, Jr. The content of the material concerned a progressive private housing project, and, although it was uniformly interesting, it did not evoke any violent emotional reaction. Of course, after repeated tests the material became boring, but, since the subject was instructed to disregard the content, this factor could have no bearing on the results. It is standard practice to discard vinylite discs after 50 reproductions because of wear, but the experimental discs were discarded after only 12 reproductions in order to eliminate any possible error from this source.

Duration of Exposures and Stepwise Increases of Intensity. The duration of exposures and stepwise increases of intensity were exactly the same as were used in the pure tone experiment. The test material was started at 100 db. above 0.0002 dynes/cm.^{2} and increased in 2-db. steps every 1.5 seconds up to 130 db. Above 130 db. the intensity steps were reduced to 1 db. Because of the limitations of the apparatus and the necessity for avoiding peak clipping at high intensity levels, the highest intensity used was 140 db. (by VU meter) above 0.0002 dynes/cm.^{2}

Sequence of Observations and Scheduling. The speech tolerance subjects were divided into three groups according to the sequence of ears tested and time intervals between sessions.

For Speceh Tolerance Group 1 the order of sequence of tests was as follows:

TEST A	Right Ear
Acuity	Threshold of detectability for speech
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
	Left Ear
Acuity	Threshold of detectability for speech
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
	Five-minute rest
TEST B	Right Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech

	Left Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
	Five-minute rest
TEST C	Right Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
	Left Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
	Five-minute rest
TEST D	Right Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
	Left Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech

In Speech Tolerance Group 1, therefore, there were four tests on each ear per session administered in the pattern RLRLRLRL. Group 1 was exposed to four sessions (I to IV) of testing at weekly intervals. Thus test IIA indicates the first test of the second session.

For Speech Tolerance Group 2 the sequence of tests was as follows:

TEST A	Right Ear
Acuity	Threshold of detectability for speech
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
TEST B	Right Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
	Five-minute rest
TEST C	Right Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech

TEST D	Right Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
TEST A	Left Ear
Acuity	Threshold of detectability for speech
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
TEST B	Left Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
	Five-minute rest
TEST C	Left Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech
TEST D	Left Ear
Discomfort	Threshold
Tickle	Threshold
Pain	Threshold
Acuity	Threshold of detectability for speech

In Speech Tolerance Group 2, therefore, there were four tests on each ear per session administered in the pattern RRRRLLLL. Group 2 was exposed to four sessions (I to IV) of testing at weekly intervals.

The difference in sequence between Groups 1 and 2 was introduced to detect the development of a "contralateral tolerance." It seemed possible that a tolerance test performd on one ear might affect the tolerance of the opposite ear by making the subjct accustomed to very loud sounds, but no such general effect was detected.

For Speech Tolerance Group 3 the sequence of tests was exactly the same as for Group 1: that is, RLRLRLRL. However, the time interval between sessions was 24 hours instead of one week. The difference in time interval between Groups 1 and 2 on the one hand and Group 3 on the other was introduced to study the persistence of after-effects of the tests of tolerance as a function of the interval between sessions.

The time consumed in an experimental session of speech tolerance generally ranged from 55 to 70 minutes.

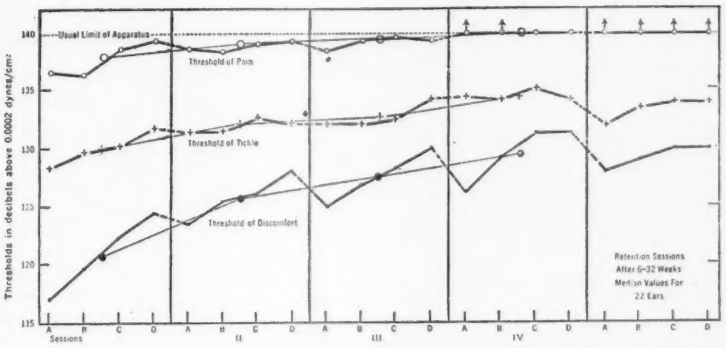

Fig. 6.—Elevation of tolerance for speech—Normals. All groups. Mean of median values for 30 ears. Heavy lines indicate means of median for each test; light lines indicate means for each session.

Thresholds of Acuity. These were determined at the beginning of each session and immediately following each discomfort-tickle-pain series. This was done to determine what effect the exposure to high-intensity speech might have on the threshold of detectability for speech. The threshold of detectability is the point at which the subject just hears the sound of speech without regard for intelligibility.

Subjects. There were 5 normal and 5 hard-of-hearing subjects in each speech tolerance group. There were thus 10 normal and 10 hard-of-hearing ears in each group or a total of 30 normal and 30 hard-of-hearing ears in the speech tolerance experiment.

RESULTS—NORMALS

Ultimate Threshold of Tolerance. The ultimate level for each threshold was derived from the asymptotes of the curves in Fig. 6 which represent the means for each session. These values were 129.5, 134.6 and greater than 139.2 db. for discomfort, tickle, and pain, respectively. In addition, the means for each test are plotted in Fig. 6 to show in greater detail the elevation of thresholds with experience.

RESULTS—HARD-OF-HEARING

Ultimate Thresholds of Tolerance. The ultimate level for each threshold was derived from the asymptotes of the curves in Fig. 7

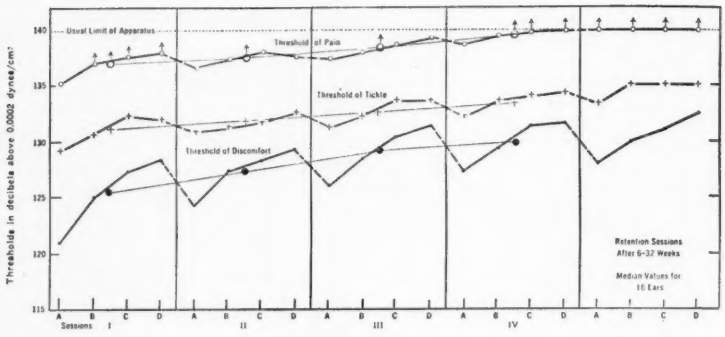

Fig. 7.—Elevation of tolerance for speech—Hard-of-Hearing. All groups. Mean of median values for 30 ears. Heavy lines indicate means of median for each test; light lines indicate means for each session.

which represent the mean for each session. These values were 130, 133.5 and greater than 137 db. for discomfort, tickle, and pain, respectively. In addition the means for each test are plotted in Fig. 7 to show in greater detail the elevation of thresholds with experience.

SUMMARY

The thresholds of discomfort, tickle, and pain produced by pure tones and speech were determined in approximately 16,000 observations on 46 normal and 46 hard-of-hearing ears, the latter representing a fairly balanced distribution of clinical types of deafness.

The initial pure tone thresholds for pain and for tickle lie at about 140 db. and 133 db. above 0.0002 dynes/cm.^{2}, respectively, for all frequencies from 250 to 5600 c.p.s. The median thresholds are higher for the normal than for the hard-of-hearing group, the more "tender" hard-of-hearing individuals reporting tickle and sometimes pain between 120 db. and 130 db.

The contour of the threshold of discomfort approximates that of an equal-loudness contour and shows a broad minimum (3 db. below the mean) between 1400 and 4000 c.p.s. The initial median threshold for the normal (110 db.) lies below the threshold for the

TABLE 1.—INITIAL AND FINAL THRESHOLDS IN NORMAL AND DEFECTIVE HEARING FOR PURE TONES AND SPEECH.

Mean of Medium Values in decibels above 0.0002 dynes/cm.^{2}

	DISCOMFORT		TICKLE		PAIN	
	INITIAL	FINAL	INITIAL	FINAL	INITIAL	FINAL
Normal Hearing						
Pure Tones	109.9	120.0	>133.1	>141.9	>140.1	>139.2
Speech	117.0	129.5	128.3	134.6	137.9	>139.2
Defective Hearing						
Pure Tones	118.4	129.5	129.4	>141.1	>136.3	>141.3
Speech	121.0	130.0	129.3	133.5	135.3	137.0

hard-of-hearing (118 db.), but there is great dispersion, particularly among the hard-of-hearing.

Similar determinations made with carefully monitored (recorded) speech gave a similar set of values (Table 1). Tickle is a more constant phenomenon for speech than for pure tones and has a lower threshold as measured by a VU meter, but pure tones cause more discomfort at lower intensities in normal ears than does speech.

The thresholds of discomfort, tickle, and pain rise systematically and significantly, with successive test sessions either daily or weekly, and approach limiting values after several test sessions.

The increased tolerance is largely but not entirely retained after an interval of a week, and more than half of the increase is retained for at least 26 weeks for the normal group and 32 weeks for the hard-of-hearing group.

Development of tolerance in one ear does not increase the corresponding tolerance in the other ear of the same individual. In the normal group exposure to speech is as effective as exposure to pure tones in elevating tolerance for pure tones. In this group the pure tone tests are only one third as effective as the original speech tests in elevating tolerance thresholds for speech. A reverse relationship exists for the hard-of-hearing.

Tolerance may be developed effectively by exposure to loud speech at a level just below the threshold of discomfort for several minutes a day at three or four weekly intervals.

Exposure sufficient to produce maximal elevation of the tolerance threshold causes at most only a small transient rise in the threshold of acuity.

GENERAL OBSERVATIONS AND DISCUSSION

After each test throughout all experiments, the subjects were asked to comment on how they felt as a result of the exposure to the tests. In about half the cases the subjects made no comment. In about 75 per cent of the comments which were made, the report mentioned a "tickling" sensation. The next most frequent comment was "buzzing" (tinnitus) and "fullness in the ear." About 3 per cent of the reports suggested "dizziness." Two subjects in early experiments on pure tones reported a feeling of nausea after the exposure. In all cases the conditions were temporary, disappearing usually a few minutes after the test. In two individuals, the feeling of "fullness" did not disappear for a few days. The subjects were usually cooperative and were probably motivated by the feeling that despite their handicaps they were making a unique contribution to the war effort.

Those subjects who were habitual hearing aid users all reported that the experimental electro-acoustic system was far superior in quality to their own hearing aids, even when the subjects were perfectly satisfied with the latter. The comment, "I wish I had a hearing aid like that" (the experimental apparatus), was practically universal. This confirms the report[1] of the Psycho-Acoustic Laboratory that a high fidelity system is most desirable in a hearing aid regardless of any theories concerning "selective amplification."

The data indicated that 130 db. appears to be the greatest useful maximum output of a hearing aid. The phenomenon of progressive elevation of tolerance with exposure to high-intensity stimuli suggests that preliminary determination of the tolerance threshold in the clinical selection of hearing aids should allow for an eventual rise in the tolerance threshold. Moreover, clinical practice should be directed toward elevating the tolerance threshold, since the high level of tolerance thresholds reached experimentally suggests that there is an approachable and potentially useful portion of the auditory area beyond the range of present audiometry. Consequently, some individuals who have heretofore been termed "totally deaf" as a result of audiometric tests might be reached by auditory stimulation through properly designed apparatus.

[1] The complete report will be available in the very near future in photostat or on microfilm from the Office of Technical Services, Department of Commerce, Washington, D. C.

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LVII

THE USE OF PLASTIC TUBING IN THE TREATMENT OF CHRONIC MAXILLARY SINUSITIS

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The treatment of chronic suppurative maxillary sinusitis is by no means ideal. The advent of chemotherapy and biotherapy has assisted immensely in the management and cure of acute and subacute suppurative sinus infections, but chronic suppurative diseases of the sinuses stubbornly remain a problem. Chronic purulent infection in the nasal cavity should be differentiated from allergic, hypertrophic, and vasomotor conditions and, quite importantly, from exaggerated physiological responses to natural environmental stimulations. The majority of so-called chronic conditions in the nose are not purely suppurative in character but are associated with nasal congestion and excessive mucous secretion on an allergic or physiologic basis.

The nature of the occurrence of this chronicity is worthy of investigation. The vast majority of cases of acute suppurative sinusitis resolve quickly. The small number that tend to become chronic are treated most satisfactorily during this early phase of their chronicity. Specific and contributory causes for the suppuration should be sought and applied. The cases which have not responded to these specific and general measures and have become chronic concern us in this paper.

Fifteen individuals with proven sinus disease of more than three months' duration were selected for treatment. Seven were identified as not possessing any contributory cause associated with the infection. Of the remaining eight, four had allergies, three of these with polyps and hypertrophic tissue. One had an associated apical abscess, and three had septal deviations of more than average degree toward the involved side. Eleven had x-ray evidence of involvement of one or more of the other nasal sinuses in addition to the maxillary sinus.

The plan was to treat these chronic infections energetically with 300,000 units of penicillin and 90 grains of sulfadiazine systemically daily for seven days and to inject penicillin, 500 units per cc., locally

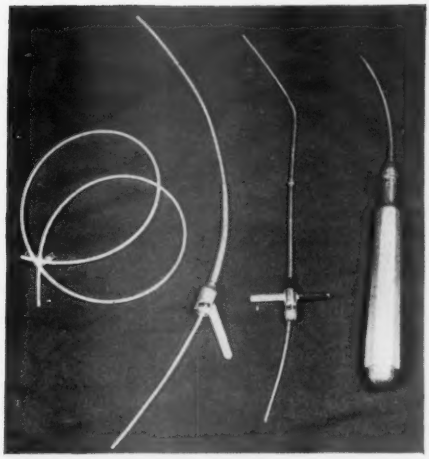

Fig. 1.—Trocars and plastic tubing used in engaging the inferior meatus.

into the sinus for from five to fifteen days. Local nasal shrinkage, removal of polyps, submucous resections, and allergic management were used as adjuncts prior to this therapy, when they were indicated. Caldwell-Luc operations were reserved for cases which completely failed to respond to all the other measures.

Lavage of the maxillary sinus in the treatment of chronic suppurative disease of the antrum has been a standard treatment for many years. Usually, sterile physiological sodium chloride solution was used as the irrigating fluid. However, more recently, sulfonamides and penicillin solutions have been utilized, but their exact value has not been established. Most cases were improved after four to six lavages. In many of the cases that showed improvement and in some that were apparently cured, there was a marked tendency for the infection to reappear with subsequent upper respiratory infections.

Several years ago, we inserted a curved metal cannula through the inferior meatus into the antrum and permitted it to remain in position for from three to five days. The sinus was then irrigated generously with sterile physiological sodium chloride solution three times a day, and sulfadiazine was given by mouth. Improvement was usually striking, but again, in the chronic cases, reinfection

Fig. 2.—Plastic tubing in position.

occurred in many within six months. In retrospect, it appeared to be a rational approach to an unhappy situation. In order to facilitate the intrasinus application of penicillin solution, it was decided to repeat the treatment in a more energetic manner. A small plastic tube was inserted directly into the antrum, and the sinus was washed as often as necessary over as long a period of time as was desired. The retention of the plastic tubing in the sinus for as long as 15 days was tolerated well by all the patients and proved to be a simplified means of administration and comfortable to the individual.

The technique employed was very similar to that used in routine maxillary sinus lavage. The inferior meatus was anesthetized with 2% pontocaine applied topically. A Coakley trocar or Lichwitz needle was inserted into the maxillary sinus. A culture of the sinus was obtained by injecting from 15 to 25 cc. of sterile physiological sodium chloride solution into the sinus cavity and then aspirating it back into the syringe. With the trocar still in position, a section of plastic tubing 0.7 to 1.5 mm. in diameter and 13 cm. in length was threaded through its lumen until approximately 5 cm. had entered the cavity of the sinus. The cannula was then carefully withdrawn while the plastic tubing was held in position with a probe or forceps. The tubing was then curved about the lobule of the nostril and fixed in position with liquid adhesive, or collodion and cotton were applied to the tubing and the ala of the nose. The sinus was then irrigated once or twice daily with buffered penicillin solution of 500 units per cc. concentration. A stilet insert into the tubing to prevent leakage after the lavage was not necessary.

The plastic tubing is a polymer of ethylene, is boilable, does not contain a plasticiser, and is nonopaque with x-ray. It may be made thinner by simply stretching and is very flexible. It is tolerated well by all body tissues.

Originally the plastic tubing was threaded over a thin, firm, curved probe which had been inserted into the natural orifice. The tubing was inserted further into the antrum, and the probe carefully removed. Increased difficulty in entering the sinus, trauma about the orifice, and interference with drainage favored the use of the inferior meatal approach.

Technical difficulties with the inferior meatal approach were rare; however, when the sinus was filled with markedly thickened mucous membrane or polyps, it was difficult to insert the plastic tubing into the free air space of the sinus. This was determined by an increased resistance encountered when the penicillin solution was injected and also by lipiodol studies. When this occurs, the tube should be withdrawn and another attempt should be made.

Five patients underwent operations. Two submucous resections were performed before the penicillin therapy was attempted. Three Caldwell-Luc operations were carried out on the patients who failed to respond to the planned treatment.

Bacteriological studies showed staphylococcus to predominate on cultures; it was present in 13 of the cases. Staphylococcus aureus appeared six times in pure culture. Five cases revealed more than one organism. The hemolytic staphylococcus aureus and nonhemolytic streptococcus possessed anaerobic cultural qualities.

An analysis of the results of this treatment serves to emphasize certain features of chronic suppurative sinusitis and penicillin therapy. Twelve patients were markedly improved within the first five days. This fact was established primarily by a return of clear penicillin solution upon repeated irrigations of the sinuses and not upon the impression of the patient.

It was necessary to carry out supplementary treatment on eight patients before the plastic tube technique with the penicillin solution was done, because they manifested abnormalities which required other specific approaches. The involved tooth of the patient with the apical abscess was extracted and he was given dental care. Four individuals with allergies underwent allergy tests and treatments. Three of these had nasal polyps. Two other patients had submucous resections for markedly deviated nasal septums. These procedures

caused some improvement in the local condition and gave the penicillin therapy its best opportunity to succeed.

Six individuals were declared cured; they were principally from the infectious group whose illnesses were not complicated by other conditions. However, of these six, it was necessary to treat again three who experienced exacerbations of the suppurative process. Six were improved but not cured, and it is highly probable that a percentage of these will experience exacerbations of their infection. Three individuals with allergy and infection failed to improve significantly with all the various types of therapy administered, and it was necessary to perform a Caldwell-Luc operation on them. Two of these were helped by the operation, and one remained about the same.

DISCUSSION

It is obvious that, in a group of patients with chronic suppurative sinusitis, there is no single curative treatment. This applies particularly to cases in which the mucous membrane lining has undergone irreversible changes and also in which there is more than one exciting factor. An attempt should be made to re-establish physiological harmony in the sinus by applying general and specific and surgical measures. Even with the multiple energetic attempts, it is often only possible to improve the condition and not to effect a cure. The combined local and systemic use of the antibiotics assists in the management of the stubborn chronic cases. From this study, it is impossible to state that the topical use of penicillin was of unusual assistance; perhaps the use of sodium chloride solution locally and sulfonamides and penicillin systemically might have given similar results.

The use of plastic tubing facilitates irrigation of the antrum by permitting the sinus to be washed with the desired solution as often as necessary with no pain to the patient and no trouble to the doctor. It also simplifies lipiodol injections. It assists in the management of these refractive cases and, perhaps, cures an additional small percentage. However, it is not a panacea for chronic suppurative disease of the sinus and cannot be substituted as an individual specific treatment when other accessory types of therapy, such as the treatment of nasal allergy, deviated nasal septum, polyps, hypertrophic tissue, and diminished resistance to infection are indicated. The effect of this plan of treatment on the sinuses, other than the maxillary, which were involved was rarely striking. The technique is worthy

of a trial in cases which are of a chronic suppurative nature and have failed to improve under general and specific measures.

SUMMARY

A new technique of effective, controlled, painless lavage of the maxillary sinus using a plastic tubing through the inferior meatus is described.

An analysis of 15 cases is given.

139 EAST 36TH STREET.

LVIII

THE CIRCULATION OF THE LABYRINTHINE FLUIDS

EXPERIMENTAL INVESTIGATIONS IN RABBITS

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Our knowledge of the physiology of the labyrinthine fluids is still very limited.

It is more or less generally agreed upon that at least the major part of the endolymph is formed in the stria vascularis of the cochlear duct in mammalia. Nothing definite, however, is known about other sources although they most probably exist.

The endolymph flows, according to Guild[7] who draws his conclusions from the results of experiments on guinea pigs, towards the basal end of the cochlear duct, through the ductus reuniens into the saccule. From there it goes into the endolymphatic sac. It leaves the membranous labyrinth by passing through the wall of the pars intermedia of the saccus into the numerous small blood vessels in this region. Guild also postulates a flow from the utricle and the semicircular canals in the same direction towards the saccus endolymphaticus. This would imply the assumption that endolymph is also formed within the utricle and the semicircular canals. According to Wittmaack[35] this formation takes place in the macula and the cristae and in the so-called atypical epithelial formations of the utricle. Wittmaack also thinks that the resorption of endolymph takes place mainly in the endolymphatic sac but that a certain part of it diffuses through the membrane of Reissner into the scala vestibuli.

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Guild expected that if his conclusions as to the circulation of the endolymph were correct, blockage of any part of this pathway would result in distention of the higher parts. Failure of the pars intermedia of the saccus to function would result in distention of the entire membranous labyrinth.

A few years later it was shown by Hallpike and Cairns[9] and later by a number of other authors, that a distention of certain parts of the membranous labyrinth, particularly of the cochlear duct and the saccule, is present in cases of true Ménière's disease. In many of the cases of Ménière's disease a fibrosis of the perisaccular tissue was seen which could be regarded as morphological evidence of a disturbed resorption of the endolymph. In some of the cases, however, the perisaccular tissue was found to be normal.[2, 17] On the other hand, a marked fibrosis was present in some normal temporal bones examined by Hallpike and Cairns. These findings make it impossible that the endolymphatic sac is the only place of resorption of the endolymph. Experimental evidence points in the same direction.

Incision, cauterization and application of pressure to the endolymphatic sac failed to cause great changes in the labyrinthine reaction of rabbits.[19] Destruction of the endolymphatic sac and the more differentiated medial portion of the endolymphatic duct (Sinus II) in monkeys did not produce any histological changes in the membranous labyrinth,[18] when examined from two to three and one-half months after operation.

The origin and ultimate fate of the perilymph has not been clarified either. Some authors, such as Hyrtl,[11] Habermann,[8] Siebenmann,[25] Herzog,[10] Fleischmann[4] and others assume that the perilymph is nothing but cerebrospinal fluid which has entered the perilymphatic spaces through the cochlear aqueduct. Others, such as Wittmaack[33] Rejtö,[24] and lately Aldred, Hallpike and Ledoux[1] believed it is derived from the endolymph which has diffused through the membrane of Reissner. Still others think that it originates from the blood in the vessels of the cochlea.

Many authors, such as Meurman,[20] Quix and v. Egmond,[23] and Jampolsky[12] assume a dual origin of the perilymphatic fluid. They believe it is derived partly from the cerebrospinal fluid and partly from certain structures of the inner ear. In the latter case it could come either from the endolymph or from the blood within the modiolus.

The differences in the refraction[26] and in the total osmotic pressure[1] between cerebrospinal fluid and perilymph make it highly improbable that the former is the only source of the perilymph. The fact that endolymph and perilymph are virtually iso-osmotic in the cat[1] favors the assumption that the endolymph gives origin to at least a part of the perilymph.

Morphological evidence, however, points to the modiolar vessels as a possible source of origin of part of the perilymph, particularly in man and monkeys.[20, 21] Contrary to the findings in most of the laboratory animals, which have a short and wide cochlear aqueduct, in man, it is much longer and narrower. In laboratory animals the structures within the modiolus are separated from the scalae by a thin layer of compact bone. Part of this bony layer is replaced by loose areolar tissue in man, through which a diffusion of fluid from the vessels in the wide vascular spaces could easily take place. Additional evidence against the assumption that the cerebrospinal fluid which has reached the inner ear through the aquaeductus cochleae is the only source of the perilymph in man is furnished by the fact that complete occlusion of the cochlear aqueduct has been observed without histological changes in the inner ear.[14, 33, 37]

Uyama[28] reports distention of the membranous labyrinth following experimental closure of the cochlear aqueduct in rabbits. Only a short abstract of his paper was available to us from which no comment can be made.

Still much less is known about the ultimate fate of the perilymphatic fluid than about its origin. The only reference in the literature which we could find was the remark of Zange[36] that resorption of perilymph may take place in the perilymphatic tissue.

In an attempt to answer the question whether the perilymph is partly or entirely derived from the cerebrospinal fluid, various substances were injected into the subarachnoid space. Their spread through the cochlear aqueduct towards the inner ear was then studied.

Many authors, beginning with Weber[30] and Weber-Liel,[31] injected stained fluids or gelatins or ink into the subarachnoid space of human cadavers and dead animals and succeeded in getting them into the scala tympani. This was taken as proof of the existence of an open communication between the two spaces. According to Jampolsky[12] the results of these experiments cannot be regarded as absolutely convincing because the physical conditions prevalent in

cadavers are different from those in the living, and conditions found after death might be different from those occurring in the living.

Another group of authors made their experiments on living animals. Most of them injected granular suspensions into the subarachnoid space, such as cinnabar,[15, 22] India ink,[12, 13, 20, 34] or animal charcoal.[20]

The results obtained in these experiments are not uniform. The majority of the authors found granules in the scala tympani and took this as evidence of an open communication between the two spaces in question. Quincke,[22] however, found among five dogs in which cinnabar emulsion was injected into the subarachnoid space of the spinal cavity only one in which the emulsion was seen in the scala tympani. Unfortunately, the cochlear aqueduct was not examined. In cats Wittmaack[34] found the aqueduct filled with ink up to its inner opening, but found no particles in the scala tympani.

Injections of granular substances, if done without increasing the intracranial pressure, are of some value for the study of the fluid exchange through the aqueduct but are unsuitable for the study of the question of where the resorption of the perilymph takes place. As Weed[32] emphasizes, the absorption of fluids follows entirely different laws than that of granular substances. In the absorption of granular substances phagocytosis, adsorption, surface tension and other physical factors play an important role. In the absorption of fluids, however, the question of diffusion through membranes is a most important consideration.

Similar objections can be made to the injection of stained gelatin (carmine-gelatin[13]) for absorption studies.

Soluble dyestuffs, such as methylene blue, fuchsine and carmine, are of value in the demonstration of the ultimate pathway of normal absorption but wholly unsatisfactory for the microscopic study of the details of the actual process of absorption because of their action as diffuse or selective tissue stains.

According to Weed[32] the most satisfactory method for the study of the absorption of the cerebrospinal fluid is the injection of a 1% solution of potassium ferrocyanide and iron-ammonium citrate in equal amounts in distilled water, which is practically isotonic with the body fluids. After completion of the injection, the brain and the meninges are fixed in a formalin solution containing 1%-5% hydrochloric acid. The resulting precipitate of Prussian blue is not

affected by the technical steps used in preparing microscopic sections in which the characteristic fine blue granular precipitate can easily be identified.

Weed was able to demonstrate that in no case in which the injections were made intravitam or in the agonal stages were there evidences of general tissue absorption. With this method the true pathway which is normally pursued in the absorption of the fluids under study can be found, for the results are obtained with fluids practically isotonic with the body fluids.

Small amounts of the one per cent solution are practically nontoxic. Higher concentrations are toxic and can only be used in agonal experiments.

Weed found that the injected fluid regularly spreads into the perilymphatic space. "This connection between subarachnoid space and internal ear has been generally accepted for years and is here mentioned merely as one of the usual findings after a subarachnoid injection of either ferrocyanide solution or carbon suspension."

Kibata[16] and Meurman[20] used the method described above for injections into the subarachnoid space. Guild[7] studied the absorption of the endolymph through injections into the cochlear duct of guinea pigs. Quix and v. Egmond[23] followed the penetration of cocaine-iron salt solutions from the middle ear into the labyrinth of guinea pigs.

Kibata's paper which is quoted by Quix and v. Egmond was not available. Meurman drilled a small hole into the promontory of one of the ears of rabbits and in this way opened the scala tympani. He then injected the ferrocyanide solution into the subarachnoid space and it appeared at the opening into the promontory after one to two minutes. On histological examination numerous granules were found within the aquaeductus cochleae on the operated side but only a few in the scala tympani of the basal coil. On the nonoperated side there were many less granules in the aqueduct and none in the scalae. In animals in which India ink had been injected without any particular increase in pressure, particles penetrated into the scalae. Meurman takes this as evidence that fluid may penetrate from the arachnoid space into the scala tympani but that this communication is not always open. Even then he feels that, in all probability, a diffusion, particularly between heterotonic solutions in the scala tympani and the subarachnoid space, easily takes place.

In some of the animals of Quix and v. Egmond a small amount of the cocaine-iron salt solution had gotten from the middle ear into the subarachnoid space. It had spread from there into the middle half of the aqueduct but had not reached the inner opening. Some granules were also found in the fundus of the internal auditory meatus and along the nerve fibers in the modiolus. The authors assume that there is an open communication between the subarachnoid and perilymphatic spaces along the perineural spaces of the branches of the acoustic nerve but not by way of the cochlear aqueduct.

Jampolsky found that India ink injected into the subarachnoid space of cats and dogs had penetrated into the spiral ganglion and along the utriculo-ampullar and saccular nerves into the subepithelial tissue of the respective sense organs. He concludes that the perineural route represents in addition to the aquaeductus cochleae route an important communication between the subarachnoid space and the perilymphatic space. In rabbits Meurman found only a small amount of India ink in the fundus of the internal auditory meatus and occasionally some granules within the modiolus between the fibers of the cochlear nerve. In his opinion these findings confirm the common notion that an open communication between the subarachnoid space and the inner ear through the internal auditory meatus does not exist and that the arachnoidea in the rabbit is rather closely attached to the trunk of the eighth nerve.

In man proof of the patency of the aquaeductus cochleae is found in the fact that after traumas or brain operations with subarachnoid hemorrhages blood penetrates, in very many instances, through the aqueduct into the scala tympani.[3, 5, 6, 21, 27, 29]

In an attempt to clarify at least some of the many controversial questions, animal experiments were performed on rabbits using the technique worked out by Weed.

It was fully realized from the very beginning that because of the considerable differences in the anatomical structure of the aqueduct, results obtained in the rabbit must not necessarily be valid for man. Nevertheless the experimental approach to the problem seemed to us the most promising procedure.

It might be advisable to give, before discussing the experiments, a description of the aquaeductus cochleae in rabbits. Our own findings are in almost complete agreement with those of Meurman.

The aquaeductus cochleae in rabbits has an average length of 2.5-3 mm. and an average width of 0.3-0.5 mm. Towards the in-

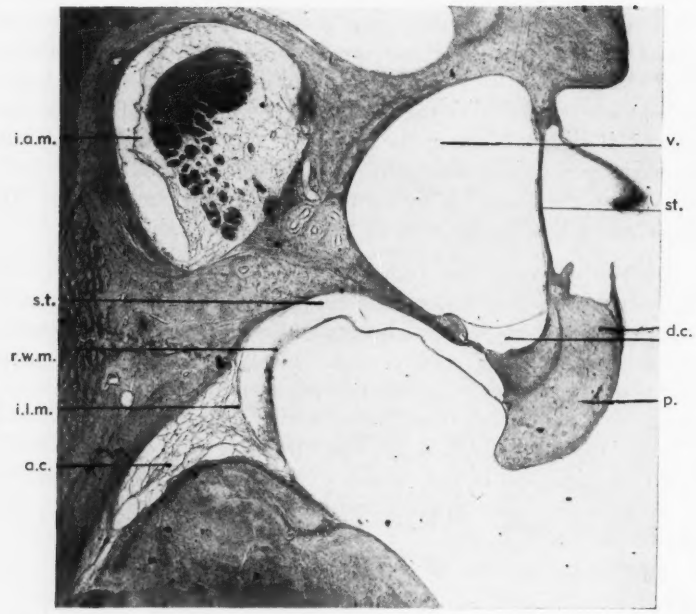

Fig. 1.—Rabbit 114; nonoperated side. Section through the cochlear opening of the aquaeductus cochleae, round window, vestibule and internal auditory meatus. *a.c.*—aquaeductus cochleae; *d.c.*—ductus cochlearis; *i.a.m.*—internal auditory meatus; *i.l.m.*—internal limiting membrane; *p.*—promontory; *r.w.m.*—round window membrane; *s.t.*—scala tympani; *st.*—stapes; *v.*—vestibule.

ternal and external openings the canal gets somewhat wider. The aqueduct usually opens into a recessus of the scala tympani which is separated from the round window niche by a lower extension of the round window membrane. This extension forms approximately a right angle with the upper part of the membrane (Fig. 1).

The aqueduct is filled with a network of loose reticular tissue which extends through the cochlear opening of the aqueduct into the recessus of the scala tympani. Towards the free lumen of the scala the reticulum forms in many places a thin "limiting membrane" (Fig. 5) but in other places such a membrane is not clearly outlined.

A comparison of the cochlear aqueduct of the rabbit with that of man shows that in the former the canal is very much wider than in the latter. Still more important is the relation between length and width. In rabbits it is 5-7:1 and in man several hundred to one. Considering this fact a communication between the perilymphatic and subarachnoid spaces in rabbits should be accomplished much easier than it is in man. The narrowness of the aqueduct in man may be compensated for by some other mechanism which is, possibly, as already mentioned, manifested by the different structure of the modiolus.

DESCRIPTION OF THE ANIMAL EXPERIMENTS

In seven rabbits (Nos. 100-104, 106, 107) 0.5-1.0 cc. of a 1%-1.66% solution of potassium ferrocyanide and iron-ammonium citrate was injected into the cerebellomedullar cisterna after withdrawal of the same amount of cerebrospinal fluid.

Three rabbits were killed after 20, 70 and 120 minutes and the temporal bones fixed in a 10%-16% solution of formalin to which 2%-5% of hydrochloric acid was added. In four rabbits 1 cc. of a 0.8%-1.0% solution of hydrochloric acid was injected into the cerebellomedullar cisterna 15 to 20 minutes after the first injection. One animal died immediately after the second injection, the others were killed 10-20 minutes later, 15, 29, 35 and 40 minutes after the first injection.

The histological examination of the temporal bones and the brain left in situ showed varying amounts of blue granules in the subarachnoid spaces and within the loose reticular tissue in the central and middle part of the aqueduct. In only two cases were granules found in the peripheral part of the aqueduct, right up to the "internal limiting membrane."

In none of these animals were granules found in the perilymphatic or endolymphatic spaces.

In four cases granules were found in the arachnoid cisterna of the fundus of the internal auditory meatus and in the perineural spaces of the acoustic and facial nerves. In three cases the granules were entirely absent in the fundus. In one case they extended for 0.1 mm. along the fibers of the cochlear nerve into the modiolus. In general the number of granules present in the fundus was definitely smaller than that in the aqueducts of the respective animals.

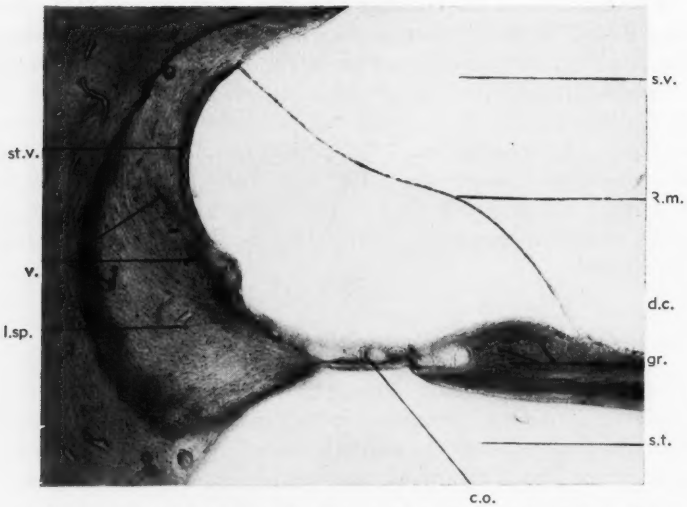

Fig. 2.—Rabbit 105. Cross section through the basal coil of the cochlea. *C.o.*—organ of Corti; *d.c.*—ductus cochlearis; *gr.*—blue granules in crista spiralis; *l.sp.*—ligamentum spirale; *R.m.*—Reissner's membrane; *s.t.*—scala tympani; *s.v.*—scala vestibuli; *st.v.*—stria vascularis; *v.*—vessels in spiral ligament filled with blue granules.

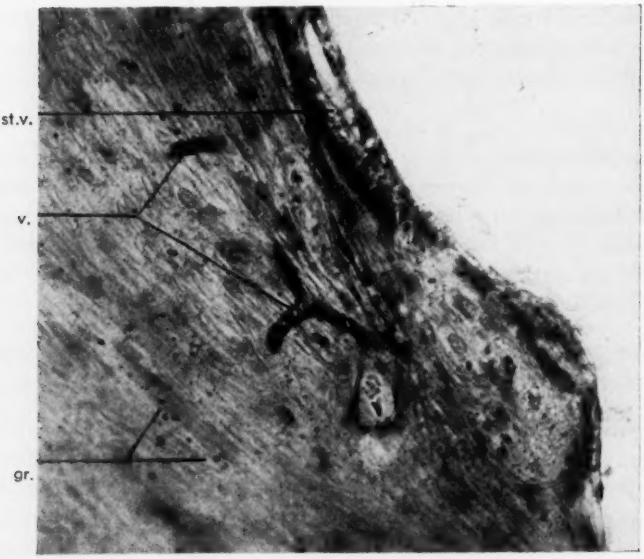

Fig. 3.—Rabbit 105. Cross section through the upper part of the spiral ligament. *gr.*—blue granules in tissue spaces; *st.v.*—stria vascularis; *v.*—vessels containing blue granules.

There was no difference between the findings in the animals which got an intravitam injection of hydrochloric acid and those which had the temporal bones fixed in the formalin-hydrochloric acid solution.

There was no clear-cut relationship between the time elapsed from the first injection to the time of death and the number of granules present, although the number was strikingly small in the animal which had lived for two hours after the injection. In this case there were a few granules in one aqueduct only. The other aqueduct and both internal auditory meatuses were entirely free of granules.

In one animal (No. 105), 1 cc. of a 5% solution of iron salts was injected; the rabbit died after nine minutes. Immediately after death 1 cc. of 1% solution of hydrochloric acid was injected into the cerebellomedullar cisterna, then the temporal bones taken out and fixed in a 40% formalin solution to which 5% hydrochloric acid had been added.

The histological examination showed the following:

Many granules were present within the subarachnoid spaces and in both aqueducts. There was a marked accumulation of granules at the cochlear opening of the aqueducts right up to the border between the reticular tissue and the free lumen of the scala tympani. An "internal limiting membrane" was visible in many but not in all the sections.

There were many granules in the scala tympani in all turns of both cochleae, but only a very few granules in the scala vestibuli. Some granules lay on the outer surface of the membrane of Reissner, others inside the cochlear duct, on the surface of the tectorial membrane and in Corti's and Nuel's spaces.

Numerous granules were found within the tissue spaces of the spiral ligament and of the crista spiralis. Many of the small thin-walled vessels in these two regions were jammed with blue granules and thus had their course and ramifications clearly outlined (Figs. 2 and 3). Other vessels contained smaller numbers of granules. A few granules only were seen within the stria vascularis. In those areas in which the lumen of the scalae, particularly the scala tympani, was lined by a thinner layer of connective tissue, the number of granules within the tissue spaces and the vessels was considerably smaller.

Fig. 4.—Rabbit 105. Section through the fundus of the internal auditory meatus and the saccular nerve. *gr.*—blue granules among the nerve fibers; *i.a.m.*—internal auditory meatus; *v.*—vestibule.

Some of the larger veins in the modiolus also contained granules together with blood elements.

There was a moderate number of granules in the loose meshwork of perilymphatic tissue of the medial parts of the perilymphatic cisterna of the vestibule, but no granules in the perilymphatic spaces of the semicircular canals.

No granules were found in the lumina of the utricle, saccule, semicircular canals, endolymphatic duct and sac. A few granules, however, were found among the epithelial elements of the sensory end-organs and more numerous granules were found in the subepithelial connective tissue and within the lumina of some small thin-walled blood vessels in this area. Even some of the veins in the deeper tissue layers also contained granules between the blood elements. The greatest number of granules was present in the macula sacculi, a somewhat smaller number in the macula of the utricle and the smallest number in the cristae ampullares.

A few granules were found in the loose connective tissue which surrounds the endolymphatic sac.

The arachnoid cisterna in the fundus of both internal auditory meatuses as well as the perineural spaces of nerves within the fundus

contained numerous granules. The granules accompanied branches of the vestibular nerve through the bone to the respective end-organs (Fig. 4). Granules were also found along the branches of the cochlear nerve within the modiolus and the lamina spiralis ossea.

No granules were seen in any parts of the osseous labyrinthine capsule or of the middle ear.

In two rabbits (Nos. 113, 114) the left middle ear was opened, the promontory exposed and a small hole made into it. Clear fluid emerged from the opening immediately after it had been made and continued to seep out throughout the experiment.

One rabbit was killed after ten minutes.

The histological examination showed that the operative hole led into both scalae of the basal coil. There was a moderate amount of fresh blood in the basal part of the scala tympani. An "internal limiting membrane" at the border between the reticular tissue and the free lumen of the scala tympani was completely absent on the operated side but was recognizable, in many of the sections, on the opposite side. Otherwise, there were no differences between the two ears.

In the second rabbit (No. 114), 2 cc. of a 1% iron salt solution were injected into the cisterna cerebellomedullaris 15 minutes after opening the promontory. One-half minute later the fluid that was seeping out of the opening in the promontory gave the Prussian blue reaction. Five minutes later the animal was killed and fixed in 10% formalin and 5% hydrochloric acid.

The histological examination showed the following:

The subarachnoid spaces contained a large amount of granules.

Left ear (operated): The reticulum of the cochlear aqueduct contained numerous granules in all parts of the duct; the "internal limiting membrane" was nowhere recognizable.

The basal parts of the scala tympani contained fresh blood and numerous granules. Granules were fairly numerous in all the other parts of the scala tympani and in the scala vestibuli. The operative opening led into the scala vestibuli of the basal coil, the membranous labyrinth being left intact. Some bone splinters were pushed into the lumen. The cochlear duct contained numerous granules, particularly on the surface of the stria vascularis and in Corti's and Nuel's spaces. Wide areas of the tectorial membrane showed a diffuse

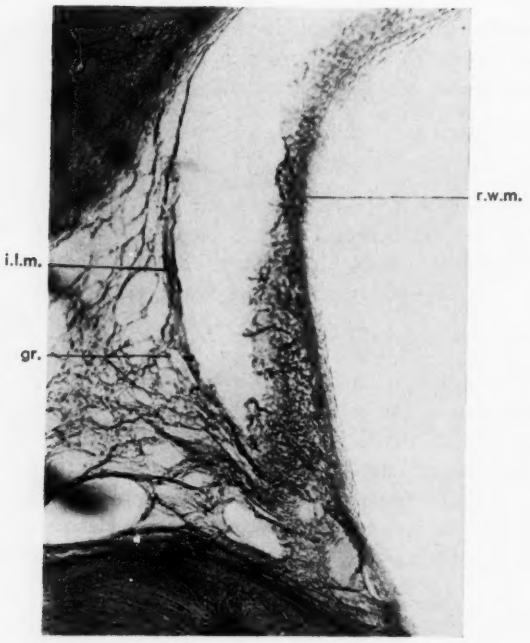

Fig. 5.—Rabbit 114; nonoperated side. Section through the cochlear opening of the aquaeductus cochleae. *gr.*—blue granules; *i.l.m.*—internal limiting membrane; *r.w.m.*—round window membrane.

blue stain. Granules were also found in the tissue spaces of the spiral ligament and of the crista spiralis, as well as in the lumina of some of the thin-walled blood vessels in these areas. There were hardly any granules within the stria vascularis. Granules were furthermore found in the lumina of some of the veins in the osseous interscalar septa.

There were some granules in the perilymphatic spaces of the vestibule and of the semicircular canals. The lumina of the saccule, utricle and semicircular canals were free of granules; some granules were seen in the subepithelial tissue of one of the cristae ampullares.

The endolymphatic duct and sac did not contain any granules but some were found in the loose perisaccular tissue.

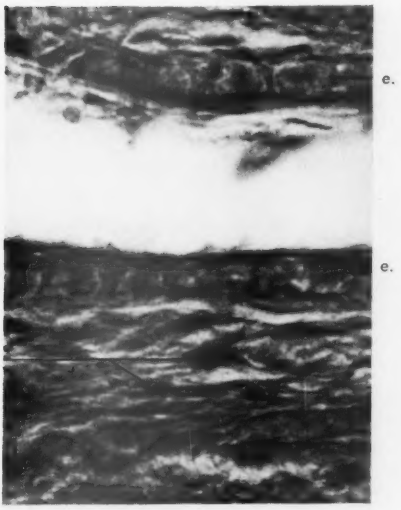

Fig. 6.—Rabbit 114; nonoperated side. Endolymphatic sac. *e.*—epithelial lining; *gr.*—blue granules in the perisaccular tissue.

Moderately numerous granules were found in the internal auditory meatus and the perineural spaces therein. No granules were seen extending along the nerves into the modiolus or the vestibule.

The osseous labyrinthine capsule and the middle ear were entirely free of granules.

Right Ear (not operated): The right aqueduct contained a considerably smaller number of granules along its entire length. An "internal limiting membrane" was recognizable in many, but not in all, sections (Fig. 5). There were a few granules precipitated on the walls of the scala tympani and the scala vestibuli. Granules were also found within the cochlear duct mainly of the basal turn, some of them within Corti's and Nuel's spaces. The tissue spaces of the spiral ligament and of the crista spiralis, chiefly of the basal turn, contained numerous granules; others filled the lumina of many of the thin-walled blood vessels in these areas. Very few granules were seen in the stria vascularis. The other turns of the cochlear duct were almost free of granules.

There were no granules in the perilymphatic or endolymphatic spaces of the vestibule and of the semicircular canals or in the sub-

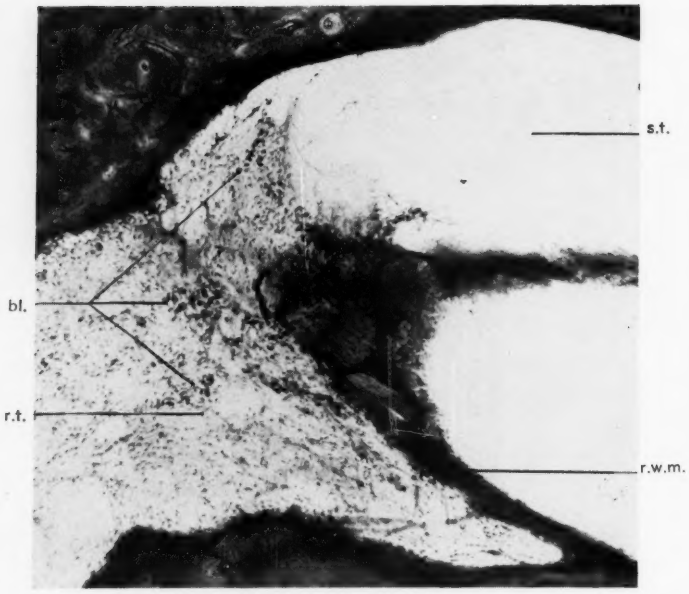

Fig. 7.—Rabbit 109. Section through the cochlear opening of the aquaeductus cochleae. *bl.*—chicken-blood cells; *r.w.m*—round window membrane; *r.t.*—reticular tissue in aquaeductus cochleae and recessus of scala tympani; *s.t.*—free lumen of scala tympani.

epithelial tissue of the sensory end-organs. The endolymphatic duct and sac did not contain granules; some of them, however, were found in the loose perisaccular tissue just underneath the epithelial lining (Fig. 6).

Only a few granules were seen in the fundus of the internal auditory meatus and the perineural spaces of the nerves therein. No granules extended along the nerves into the modiolus or the vestibule.

The osseous labyrinthine capsule and the middle ear were entirely free of granules.

In two rabbits (Nos. 108, 109) after withdrawal of spinal fluid, 1 cc. of oxalated chicken blood was injected into the cerebellomedullar cisterna. Both animals had convulsions and rapid breathing after

the injection but later recovered. One was killed 90 minutes, the other one 95 minutes after the injection.

In both animals large amounts of chicken blood (recognizable by the nucleated erythrocytes) were present in the subarachnoid spaces and in both cochlear aqueducts. In Rabbit 108 the blood extended on one side from the aqueduct into the scala tympani of the basal coil. On the other side the amount of blood was smaller and reached only to the border between the reticular tissue and the free lumen of the scala tympani, but there were no chicken blood cells in the lumen. Likewise in Rabbit 109 large amounts of chicken blood extended into the scala tympani of the basal coil on one side and smaller amounts on the other side extended just to the border between the reticular tissue and free lumen. A few chicken blood cells, however, had already penetrated into the adjacent parts of the scala. An "internal limiting membrane" was visible in some places and missing in others (Fig. 7).

In one rabbit a large, and in the other one a smaller amount of blood was present in the fundus of the internal auditory meatus. Nowhere had the blood penetrated into the modiolus or the vestibule. All the other parts of the inner ear were free of chicken blood. The perilymphatic and endolymphatic spaces in both cases contained fine granular precipitations.

COMMENT

In all rabbits into which iron salt solutions were injected, blue granules were found in the subarachnoid spaces as well as in the cochlear aqueducts. Likewise in the two rabbits into which oxalated chicken blood was injected, blood cells were similarly found in the same locations. This can be explained by postulating the presence of a fluid current from the subarachnoid space towards the aqueduct. The presence of granules and blood cells in the arachnoid cisterna of the fundus of the internal auditory meatus and in the perineural spaces of the seventh and eighth nerves (although in smaller numbers) in all but three of the rabbits suggests another but weaker current travelling from the subarachnoid space to the fundus.

The findings in seven rabbits in which the granules did not extend beyond the reticular tissue within the cochlear aqueduct and the lower recessus of the scala tympani seemed at first to point to the presence of a barrier, either anatomical or functional, at the border between the reticular tissue and the free lumen of the scala tympani.

The assumption of a complete anatomical barrier is refuted by the fact that the "internal limiting membrane" shows variations in the degree of its development and did not form in any of the cases a continuous membrane demonstrable in every section.

The assumption of a functional barrier, at least for true solutions, became untenable after examinations of Rabbit 105 which died nine minutes after injection of a five per cent solution of iron salts into the subarachnoid spaces and whose inner ear definitely contained numerous granules.

The accumulation of granules in the reticular tissue at the border between the aqueduct and the free lumen of the scala tympani indicates a slowdown of the speed of the fluid current in this area but not a complete blockage of the penetration of soluble components of the spinal fluid into the scala.

There were two possible explanations for the penetration of the iron salt solution into the inner ear in this case (Rabbit 105). It could have been due to the concentration of the solution which was higher than in the previous cases. Spread by simple diffusion, however, would not account for the presence of granules in certain areas only (spiral ligament and crista spiralis) and particularly not in the blood vessels within or leading from these particular regions. Another possible explanation seems to be presented by the fact that the animal lived for a shorter time after the injection than did the other rabbits.

The latter assumption was confirmed through the findings in Rabbit 114, in which, after a hole was made into the promontory, a one per cent solution was injected and the animal killed five minutes later. Disregarding the findings on the operated side, the fact that blue granules were present in the cavities of the inner ear on the nonoperated side favors the assumption that the presence or absence of granules in the inner ear spaces depends mainly upon the time which has elapsed between the injection of the solution and the death of the animal. Evidently the solution penetrates into the inner ear in a very short time, in less than five minutes, and disappears from the inner ear spaces very rapidly, so that no granules can be found 15 minutes after the injection.

The passage of iron salt solutions from the aqueduct into the scala tympani seems to take place in areas devoid of a "limiting membrane" through the meshes of the reticular tissue and in the other areas by diffusion through the membrane itself.

In the two cases in which a hole had been made into the promontory, no "limiting membrane" at all was visible on the operated side. It is conceivable that it had been ruptured by the cerebrospinal fluid which pressed against the membrane after escape of perilymphatic fluid through the hole in the promontory. An increase in the intracranial pressure, as Meurman suspects, is not necessary for the production of this rupture.

Injection of chicken blood in the rabbits showed that a large amount of blood had penetrated into the scala tympani on one side in each rabbit after the "limiting membrane" had been ruptured. On the other side a much smaller amount of blood had entered the cochlear aqueduct but had been stopped at the border between the reticular tissue and the scala tympani in one case, while in the other case a few red blood cells were seen passing through the meshes of the reticular tissue into the free lumen of the scala tympani.

The differences in the amount of blood in the two aqueducts in the same animal are difficult to explain. There might be differences in the texture of the reticular tissue within the aqueduct or in the degree of development or extent of the "internal limiting membrane." In addition, the chicken blood exerts an irritating influence on the meninges as well as on the inner ear. The fine granular precipitations in the inner ear cavities in both cases seem to represent the first signs of a serous labyrinthitis which were completely missing in all the cases in which iron salts had been injected. It is conceivable that these changes might also have helped to create differences in the reactions of the aqueducts.

The spread of granules in one case (Rabbit 105) along the branches of the acoustic nerve into the modiolus and the vestibule and in another case (Rabbit 101) for a very short distance along the fibers of the cochlear nerve into the modiolus suggests the existence of another although weaker current from the fundus of the internal auditory meatus toward the inner ear spaces.

The findings in Rabbits 105 and 114 indicate the manner in which the iron salt solutions which had penetrated into the inner ear are eliminated.

After having entered the scala tympani of the basal turn the iron salts go up to the helicotrema and from there down through the scala vestibuli into the vestibule. From the scala vestibuli they diffuse through Reissner's membrane into the cochlear duct. There is no definite proof for or against diffusion from the scala tympani

through the basilar membrane, although the presence of granules in Corti's and Nuel's spaces could be interpreted as indication of this fact. From the cochlear duct the salts flow down through the ductus reuniens into the saccule. However, the main part of the solution is resorbed within the cochlea so that only relatively small amounts reach the vestibule.

The resorption within the cochlea occurs in two places, i.e., in the spiral ligament and its adjacent areas and in the crista spiralis. In these two areas granules are seen in the tissue spaces as well as in many of the thin-walled blood vessels which carry the fluid into the venous system. The stria vascularis seems to participate to only a very limited extent in the resorption of the fluid.

If a highly concentrated iron salt solution has entered the labyrinth, resorption is seen in all parts of the cochlea, otherwise it is observed mainly in the basal turn.

The findings in the other parts of the inner ear are more difficult to explain.

The presence of a few granules in the loose connective tissue which surrounds the endolymphatic sac seems to indicate that some of the fluid has reached the sac and has penetrated from there into the surrounding tissue.

The granules found in the subepithelial tissue of the sensory end-organs, in the lumina of some of the small veins in this area, and occasionally between the covering epithelial cells could indicate that iron salts are being resorbed from the endolymphatic spaces. However, the possibility that the salts had been carried in an opposite direction from the fundus of the internal auditory meatus along the perineural spaces of the branches of the vestibular nerve to the subepithelial tissue of the sensory nerve endings cannot be excluded. If the findings in Rabbit 105 are interpreted in this way, then it can be assumed that the salts were just about to enter the lumen of the membranous labyrinth.

The results of the experiments lead to the conclusion that the aquaeductus cochleae represents the main route for the penetration of iron salt solutions from the subarachnoid into the perilymphatic space. Aside from that, another but much less important pathway seems to exist along the perineural sheaths of the branches of the acoustic nerve. In this sense we are unable to agree with Meurman, who says that the connection through the internal auditory meatus does not exist in the rabbit. Our findings confirm, to some extent, those of Jampolsky in cats and dogs and those of other authors.

Our experiments demonstrate that iron salt solutions penetrate quite easily into the cochlear duct through Reissner's membrane and possibly also through the basilar membrane.

The resorption of the iron salts occurs not only with the perilymph in the two scalae but also with the endolymph in the ductus cochlearis and in other parts of the endolymphatic system. Although we find evidence that a small amount of the iron salts gets into the endolymphatic system and is resorbed from the endolymphatic sac, we believe that the main part of the fluid is resorbed within the cochlea. This might explain why Lindsay[18] did not find any changes in the membranous labyrinths of monkeys after obliteration of the endolymphatic sac.

Guild[7] injected iron salts directly into the cochlear duct of guinea pigs and studied the distribution of the blue granules. The perilymphatic space had also been opened in all but one animal and the injected fluid had entered this space. In 16 out of 20 cases granules were present in the wall of the pars intermedia of the endolymphatic sac. In six cases either the entire, or at least large parts, of the spiral ligament were infiltrated with blue granules. In three of the cases granules were also found about the veins leading from the ligament and in one case were present within the lumina of some of these veins. The stria vascularis was infiltrated in one case only. Guild considers the possibility that the iron salt solution was inadvertently injected directly into the spiral ligament itself. He thinks that there is not sufficient evidence that this represents a normal pathway for the escape of perilymph. In two cases granules were present in small parts of Corti's and Nuel's spaces. He explains this as being due to injury to the organ of Corti by the tip of the cannula. The tissues of the limbus spiralis were infiltrated in one animal. In two cases there were some blue granules near the nerve fibers in the lamina spiralis ossea and in the spiral ganglion, but these granules were so frequently associated with small vessels that Guild interprets it as a probable perivascular infiltration from the perilymphatic spaces into which much of the injected fluid had passed in these particular labyrinths. In four cases granules were found in one part of the wall of the saccule.

In our two cases (Rabbit 105, unoperated side of Rabbit 114) trauma cannot be held responsible for the infiltration of the spiral ligament or for the presence of granules in Corti's and Nuel's spaces, because no manipulation of any nature had been done near the labyrinth. We feel that the presence of granules in the spiral ligament

and the crista spiralis and in some of the veins in this area shows that these structures represent areas in which fluid is resorbed under normal conditions.

The scarcity of granules around the endolymphatic sac in our cases is most probably due to the fact that the animals were killed five and nine minutes after injection of the fluid into the subarachnoid space. Guild's animals, however, were killed 45 minutes to 44 hours after injection directly into the cochlear duct. In our cases only a small amount of iron salts penetrated into the endolymphatic duct. In Guild's cases the presence of granules in the lumen of the endolymphatic sac and the perisaccular tissue a long time after the injection could also indicate that the resorption in this area occurs at a much slower pace than in the cochlea.

Difficult to interpret in many respects are the findings of Quix and v. Egmond.[23] These authors injected a five per cent cocaine and two per cent iron salt solution into the middle ear of guinea pigs and studied its penetration into the inner ear. The fluid which had gotten into the two scalae of the cochlea penetrated very easily through Reissner's membrane into the cochlear duct. It was resorbed, according to the authors, into the lymph spaces of the spiral ligament and from there into the lymph spaces of the modiolus which surrounded the big veins in this area. From there the fluid went to the base of the skull via the lymphatics. The resorption occurred within a relatively short period of time. In the vestibule and the semicircular canals there was evidence of a fluid current from the perilymphatic spaces along the nerve fibers into the subepithelial layers of the tissues of the maculae and cristae. In the modiolus the authors assumed the presence of a current along the fibers of the cochlear nerve towards the organ of Corti. No granules were found in the endolymphatic duct and sac nor in the perisaccular tissue. The authors found it conceivable to assume that the endolymph and the perilymph were secreted partly by the stria vascularis and derived partly from the cerebrospinal fluid which reached the cavities of the inner ear along the fibers of the acoustic nerve. They assumed that the aquaeductus cochleae did not transmit fluid from the subarachnoid space into the scala tympani.

The importance of the ligamentum spirale for the resorption of the intralabyrinthine fluids is confirmed by our own findings. We cannot, however, agree with Quix and v. Egmond that the fluid is carried away in the lymphatics. In the first place, the existence of true lymphatics in the inner ear has never been proven. In ad-

dition, we found granules, at times along with blood, in many vessels which were unquestionably venous.

In view of Guild's results in guinea pigs, the complete absence of granules in the endolymphatic duct and sac and in the perisaccular tissue is hardly understandable in Quix's and v. Egmond's experiments on guinea pigs.

SUMMARY AND CONCLUSIONS

The wide and relatively short aquaeductus cochleae in rabbits is filled with loose reticular tissue which extends for a short distance into the adjacent recessus of the scala tympani. The reticular tissue at the border which is in relation to the free lumen of the scala tympani shows, in many places, a membrane-like condensation, which could be called an "internal limiting membrane," without forming, however, a continuous membrane in all consecutive sections.

Iron salt solutions injected into the subarachnoid space are carried by a strong current into the aqueduct and by a less active current into the fundus of the internal auditory meatus. Near the inner opening of the aquaeductus cochleae the current is slowed down, but nevertheless the solution penetrates into the scala tympani within a few minutes either through diffusion through the internal limiting membrane or through the meshes of the reticular tissue in places where the limiting membrane is defective. From the scala tympani the solution is carried upward to the helicotrema and downward through the scala vestibuli into the vestibule. At the same time it penetrates through Reissner's membrane and possibly also through the basilar membrane into the cochlear duct. A small amount of the solution is carried down into the saccule and from there into the endolymphatic sac. The greatest part of the solution is resorbed very rapidly within the cochlea. It is resorbed from the scalae as well as from the cochlear duct and it goes into the tissue spaces of the spiral ligament and the crista spiralis and from there into the venous system. A small amount of the solution is resorbed through the wall of the endolymphatic sac.

Because of the probable existence of a fluid current from the fundus of the internal auditory meatus along the perineural spaces of the branches of the cochlear and vestibular nerves into the subepithelial tissue of the sensory end-organs, no definite conclusion can be drawn about the fate of the solution which reaches the semicircular canals.

Corpuscular elements, such as blood cells, penetrating into the aquaeductus cochleae are stopped near the inner opening. Later on they may pass into the free lumen of the scala tympani either at the places where the internal limiting membrane is defective or, if they accumulate in large amounts, after rupturing the membrane.

Extreme caution seems imperative if one tries to draw conclusions, from the results of these experiments, regarding the normal physiology of the perilymphatic fluid in rabbits. In the first place the number of animals is too small and, therefore, the results obtained can only be regarded as preliminary. Furthermore, one should be aware of the fact that the fluid exchange takes place through semipermeable membranes and the fluids physiologically involved are not simple, true solutions as in our experiments.

With these reservations in mind we come to the following conclusions:

The perilymph in rabbits is mainly derived from the cerebrospinal liquor which reaches the perilymphatic spaces chiefly through the aquaeductus cochleae and to some extent also through the internal auditory meatus. In view of the fact that Reissner's membrane is so easily penetrated by iron salt solutions, the possibility exists that some of the perilymph is derived from the endolymph. In the experimental animals, however, the iron salts penetrated from the perilymphatic into the endolymphatic space of the cochlea. If the pathway which is followed by the iron salts is indicative of the physiological currents within the perilymph, then it can be stated that the resorption of the perilymph within the cochlea takes place at a very rapid pace in the ligamentum spirale and the crista spiralis. No conclusions can be drawn regarding the fate of the perilymph in other parts of the labyrinth. The same evidence makes it probable that resorption of endolymph also occurs at the same places within the cochlea. This would explain the lack of dilatation of the endolymphatic system after experimental obliteration of the endolymphatic sac.

In view of the great differences in the structure of the aquaeductus cochleae and the modiolus in rabbits and man, conclusions drawn from experiments in rabbits are not necessarily valid in man. More valuable in this respect, most probably, will be experiments in monkeys in which the structures concerned are very similar to those in man.

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LIX

CARCINOMA OF THE MIDDLE EAR AND MASTOID PROCESS

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Cancer of the ear, in our opinion, should be classified into three groups: (1) primary carcinoma of the external ear canal and auricle (extrinsic), (2) primary carcinoma of the middle ear and mastoid process (intrinsic), and (3) neoplasms involving the middle ear, the mastoid process, the external ear canal and the auricle whose site of origin is impossible to determine at first examination.

The purpose of the paper will be to add to the literature six cases of carcinoma of the middle ear and mastoid process and to discuss some of the more important clinical aspects associated with this disease. It is noteworthy that in not a single instance were we able to prove conclusively that the patient had an authentic primary carcinoma of the middle ear and mastoid process; that is, they all were classified as tumors of the third group.

Leading otologists[1-4] have adequately revealed how seldom a bona fide intrinsic (primary) carcinoma of the middle ear is seen. Furstenberg[5] made an exhaustive study of the literature in 1924 and he could find a total of only 75 authenticated cases of primary carcinoma of the tympanum, most of which were culled from the foreign literature. He deduced the incidence as being 1 to every 20,000 otological conditions.

Regarding the etiology of these neoplasms, Figi and Hempstead[6] tend somewhat to minimize chronic otorrhea as a strong causative factor. They state that in only 11 of their 38 treated cases was there a history of long continued aural discharge. In our

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series of six cases, there was chronic otorrhea for over 23 years in three. One patient had chronic otorrhea for seven years, one for five months and the other had no previous discharge at all.

It is noteworthy that all six cases in our series occurred in white females. These figures differ from those of Schall,[7] who reported on six cases in 1935, three being in each sex.

Bilancioni[8] observed sarcoma of the middle ear in a child of five years. Alexander[9] reported a case which he saw in a boy of 17. In our series of six cases, the youngest patient was 25 years old, the oldest 68 years. Sixty-six per cent occurred between the ages of 40 and 70 and approximately 50 per cent between 50 and 70.

It is difficult to determine the exact site of origin of carcinoma of the middle ear since the disease usually is well advanced at the time it is discovered. Some authorities feel that the process starts deep in the external auditory canal or in the tympanic membrane and then invades the middle ear and mastoid process; others, chief among whom is Barnes,[10] feel that a large number develop from the lining of the mastoid cells which have become altered during a chronic suppurative process. These last types almost invariably produce late symptoms, which are usually pain and facial paralysis.

Lederer[11] offers the membrana tympani as a natural anatomical delineation for classifying "extrinsic" and "intrinsic" lesions of the ear. He feels that all lesions originating external to the tympanic membrane are "extrinsic lesions." These include all lesions originating in the external auditory canal whether or not they begin "in the area" of the drum membrane or medial to the isthmus of the external auditory canal. This seems most practical and certainly would tend to resolve any confusion as to just what constitutes an "intrinsic" (primary) lesion and just where an intrinsic lesion ends and an extrinsic lesion begins within the external auditory canal.

The most common symptoms associated with carcinomas of the middle ear and mastoid are:

1. Otorrhea, which is usually of long standing and for which the patient has most commonly been treated with various agents such as irrigations, cauterizations, and drops.

2. Pain, which is usually severe and out of all proportion to the clinical findings and which is in most instances one of the earliest warning signals for the presence of a neoplasm. Furstenberg[5] believes that pain is actually a late symptom and is due to the pressure

of the growth in the middle ear. Barnes[10] suggests that the pain is due to a secondary infection of the tumor mass and of the skin lining the external auditory canal.

3. Bleeding and ulcerations. These are often associated with granulation tissue and polyp formations.

4. Loss of hearing. However, in many instances the patient does not complain of this. In this connection it is of interest that the labyrinthine capsule usually is invulnerable to malignant invasion and rarely is disturbed until the very late stages, and then only after most of the petrous portion of the temporal bone has been destroyed by the neoplasm.

5. Facial paralysis, which is due to the involvement of the facial nerve and is usually a late symptom. However, as Barnes[10] described, if the lesion actually begins in the mastoid, this may be one of the early symptoms. In our series all six patients at one time or another developed facial paralysis, three of them presenting themselves with this symptom at the time of their first admission. All of them were later found to have neoplasms which were far advanced clinically.

6. Vertigo. This is a rare complaint.

7. Dysphagia and inarticulate speech, which suggest that the condition has already become extensive and that the ninth, tenth, eleventh and twelfth nerves in the area of the jugular bulb have become involved, giving rise to well-established syndromes associated with lesions in this area. With regard to these syndromes, it is interesting to observe their descriptions in the literature under different names. Lederer,[11] in his book, points out the futility of trying to associate specific lesions in the area of the jugular foramen with the different names attributed to them by the different authors. He points out that these lesions are usually progressive and that the lesion can change from "one syndrome" into "another syndrome" in a matter of days or even hours. He offers the only serviceable classification, basing it on the anatomical situation (Table 1). Anatomically, if the sympathetic chain is involved, an associated Horner's syndrome with ptosis, miosis, anhidrosis and enophthalmos will result. If the glossopharyngeal nerve is involved, there is a resultant loss in taste and sensation over the posterior third of the tongue with embarrassment to deglutition. If the tenth nerve is destroyed by the neoplasm, there is produced a unilateral paralysis of the pharyngeal constrictor muscles, resulting in dysphagia and unilateral par-

TABLE 1.

ANATOMIC (ASSOCIATED VOCAL CORD PARALYSIS) CONSIDERATIONS. ANATOMIC SITES FOR THE LESIONS WHICH CAUSE THE VARIOUS ASSOCIATED PARALYSES (LEDERER[11] AFTER BURGER).

SYNDROME	PARALYSES IN DOMAIN OF NERVES	VAGUS LESION
A. Bulbar	IX, X, XI, XII (V, VI, VII, VIII)	High vagus paralysis
B. Jugular foramen	IX, X, XI, XII Sympathetic	
C. Parapharyngeal space	IX, X, XII, (VII) Sympathetic	Deep vagus paralysis
D. Arch of aorta	X, phrenic Sympathetic	Recurrent laryngeal nerve or deep vagus paralysis

alysis of the vocal cord with anesthesia of the larynx on the involved side. The soft palate will be displaced toward the uninvolved side. If the eleventh nerve is infiltrated, a homolateral weakness of the sternocleidomastoid and trapezius muscles may result. And finally, when the twelfth nerve is invaded, the tongue will be "pushed" toward the diseased side.

The above anatomical description in our opinion seems to be the best way to describe lesions (in part or in their entirety) in the area of the jugular foramen. This seems more practical than to try to describe them by some name attached to a syndrome which at best could be only part of a larger picture that might develop at a later stage in the disease process.

The diagnosis of primary carcinoma of the middle ear and mastoid process must be made early if a good result is to be expected. In this connection the roentgenographic studies alone are not reliable. X-ray studies usually will suggest a neoplastic change only when it is already too late to treat the patient adequately.

If we are to achieve the required early diagnosis, it is by microscopy. Lederer[11] states that, "Histologic examination of suspicious granulation tissue, especially that which does not respond to cauterization and has a tendency to recur after removal, is important for diagnosis." This quotation is the keynote to the problem of exposing the disease early enough to allow effective therapeutic measures to be instituted.

In our series of cases all were squamous cell carcinomas. Others have described basal cell carcinomas, adenocarcinomas, and hemangioendotheliomas.

The most common route for the spread of the neoplasm seems to be by direct extension along the external auditory canal into or from the tympanum, followed by further extension to the surrounding areas, such as the temporomandibular joint, the parotid gland and the adjoining tissues as well as the cranial cavity. It is noteworthy that the dura mater is quite resistant to neoplastic invasion and is usually involved in the very late stages of the disease process. Regional lymph node metastasis occurs rather late in the disease. This is usually by extension of the disease directly to the nodes, and less often by process of metastatic emboli such as, for example, is found in the spread of carcinomas of the lip, or in the extension of nasopharyngeal fibromas.

Furstenberg[5] in 1924 and Schall[7] in 1935 stressed the need for radical treatment of carcinomas of the middle ear and mastoid process. Schall divided his approach into three stages: First, he effected a wide excision of all growths found in the external auditory canal medial to the isthmus, removing the cutaneous canal completely during this stage. He then did a radical mastoidectomy in order to inspect the middle ear and mastoid. This also formed a space for proper application of the radium (which was his third stage) in the treatment of the neoplasm. All these steps are carried out as a single operative procedure. Third, he applied radium in platinum needles with walls 0.5 mm. in thickness, protected by 0.5 cm. to 1.0 cm. of gauze. A total dosage of 600 to 1000 milligram hours was administered, varying with the extent of the lesion. This dosage did not usually cause cerebral damage or serious bone necrosis. Further follow-up treatments were rendered as required.

Figi and Hempstead,[6] in 1943, outlined their radical approach to the problem. Basically, they are in agreement with the principles mentioned above; they differ only in certain minor details. The method is essentially the same as that used today in dealing with malignant neoplasms of the larynx and nasal accessory sinuses. They attribute a great deal of the success of their treatment to the employment of electrosurgical methods and to more caustic doses of radium, as well as to the other principles previously mentioned. They remove the accessible portion of the neoplasm by means of electrocauterization rather than by excision. The mastoid process is then opened and, if the neoplasm is not present, the posterior wall of the

canal is removed and a radical mastoidectomy is done. If neoplastic tissue is found on removal of the bony cortex (and this is often the case), this tissue is destroyed with diathermy electrode, the dissection of bone being continued to expose the remainder of the neoplasm. In this fashion, removal of bone and electrocoagulation are alternated until the tumor has been entirely destroyed, if this be possible. During this phase large portions of the dura and the lateral sinus, or the jugular bulb and other intracranial structures may be exposed. There may be malignant infiltration into these parts. If this is the case complete removal of the growth is impossible. More often a thin capsule will be found separating the tumor from the dura and the lateral sinus and now, provided that the remainder of the growth can be completely removed, the primary one can be entirely eradicated. Furthermore, as the bony posterior wall of the external auditory canal is removed, the part of the tumor which could not at first be reached via the meatus is now accessible.

Provided the dura is intact, amazingly little postoperative reaction follows the electrosurgical procedure even though the neoplasm has been extensive and has required a great deal of electrocoagulation. After surgical excision of the growth, the patient will realize great relief from pain and aural discharge, but, if only a part of the growth has been excised, this improvement will be only temporary. The pain and the otorrhea will recur almost at once, especially if the neoplasm has infiltrated the dura. This will occur in spite of intensive irradiation which is used to supplement the incomplete surgical removal of the tumor mass.

After surgical removal of a neoplasm, radium points containing 1 mg. each of radium encased in platinum are employed. Ten to fifteen points are laid into the mastoid wound, encased in iodoform gauze which keeps them well removed from the bone itself. A total dose of 300 to 400 milligram hours are administered. A few days later, this treatment is supplemented (with radium packs) by an additional dose of 4,000 to 18,000 milligram hours. A great deal of bone necrosis will result from electrocoagulation and intensive irradiation. The resulting sequestra separate slowly and it may be a year or longer before they can be lifted out. It is not advisable to attempt to remove sequestra before they have loosened. If separation has been completed spontaneously, the process will be found well walled off and epithelization of the wound will occur rapidly, even though dura may be widely exposed. Should there be a suggestion that a recurrence of the neoplasm is present at any time after operation, then removal of the sequestrum and a biopsy may

become necessary, even though the dead bone is fixed firmly. In this instance we may be inviting a severe acute inflammatory reaction with extension of the necrosis and even meningitis. However, today, with the use of antibiotics and chemotherapeutic agents this danger is minimal.

The results in our series of six cases seem to prove that early diagnosis is essential. All our patients presented themselves at a far advanced stage in the disease process. Consequently, no matter what therapeutic measures were attempted, all ended in failures within two years after treatment. More encouraging are the results reported from other clinics. Figi and Hempstead,[6] for example, reported on 38 patients, 20 of whom were alive after two years, 15 alive after three years, 10 after seven years and 1 after 15 years. In their series it is also interesting that six patients appeared for treatment with extensive lesions late in the disease process. All six succumbed to the cancer within a few months to four years. Treatment in these cases was restricted to palliative measures.

The prognosis should be deferred at least until the mastoid process has been opened and the extent of the lesion determined. Furthermore, consideration must also be given to the grade of malignancy of the tumor and the presence or absence of regional lymph node involvement.

REPORT OF A CASE

Table 2 is a short resume of the clinical features of our six patients. Case "A. L." presented some interesting features and for this reason is described in more detail:

Mrs. A. L., a 68-year-old, white widow, was admitted to the otolaryngological ward of the Research and Educational Hospitals in a wheelchair on August 2, 1946. Her chief complaints were intermittent discharge and pain in the right ear for the past 25 years, facial paralysis since April 6, 1946, and continuous pain and discharge from the right ear since a right simple mastoidectomy which was performed on April 7, 1946.

For a period of approximately 23 years, she experienced recurring discharge from the right ear, which she treated with home remedies. Sixteen months prior to admission she began to experience pain over the right mastoid tip and right suboccipital area. This pain became more or less constant. About eight months later, she saw a doctor for the first time and was hospitalized for pain in the

TABLE 2.

SUMMARY OF CLINICAL FINDINGS IN SIX CASES OF CANCER OF THE MIDDLE EAR AND MASTOID PROCESS

DATE OF ADMISSION	AGE	SEX	CHIEF SYMPTOMS	DIAGNOSIS	TREATMENT	RESULT
8-2-46 A. L.	69	F	Otorrhea and earache 25 yrs., facial paralysis 4 mos.	Squamous cell carcinoma (Fig. 4)	Exploratory radical mastoidectomy; operation limited by extent of lesion	Expired 9-2-46
11-15-39 H. H.	25	F	Otorrhea 23 yrs.; earache 1 yr.; facial paralysis 3 mos.	Squamous cell carcinoma with hornification	Deep x-ray therapy; lesion too far advanced for further treatment	Expired 2-10-40
8-27-40 B. R.	48	F	Otorrhea 25 yrs.	Squamous cell carcinoma	Limited excision of tumor with electrocautery due to excessive bleeding at operation. Deep x-ray	Expired ?
1-24-40 M. Simp.	51	F	Otorrhea 5 mos.; pain 5 mos.; facial paralysis 1½ mos.	Squamous cell carcinoma	No treatment attempted; lesion too far advanced	Expired 3-22-40
1-24-40 M. S.	49	F	Pain right ear 4 yrs.	Squamous cell carcinoma	Radical mastoidectomy; deep x-ray therapy	Readmitted 4-15-41 with cerebral metastasis. Died shortly afterward.
11-27-39 J. C.	33	F	Otorrhea 7 yrs.	Squamous cell carcinoma	Limited excision of mass in membranous canal and parotid area	Expired 12-22-39

Fig. 1.—The external auditory meatus is completely obstructed by a dark red polypoid mass which is not ulcerated.

Fig. 2.—Admission photograph which illustrates the patient attempting to elevate the right angle of her mouth. Note loss of the right nasal labial fold and the widening of the right palpebral fissure indicative of a right facial paralysis.

Fig. 3.—Roentgenogram demonstrating the destruction of the petrosa, throwing into relief the uninvolved bony labyrinth.

suboccipital area and a diagnosis of "arithritis" was entertained. She was treated by diathermy and hyperextension of the neck via "halters." Since that time (January 1946) she has been a semi-invalid and has spent most of her time in bed. She now permitted an otologist, for the first time, to examine her and suggest treatment.

Quite suddenly and without any premonition she awoke on April 6, 1946, with a right peripheral facial paralysis. X-ray films at this time revealed: "tumor formation of the mastoid bone—this appears as a cholesteatoma." The next day, a simple mastoidectomy was performed by her surgeon with the hope that some of the "pressure" around the facial nerve would be released. Since this operation, there has been no improvement of the facial paralysis. Two months later, in June 1946, the family noticed that the patient developed an unsteady gait, which persisted up to the time of her admission. This was soon accompanied by an intense pain over the postauricular incision which became quite swollen. The right ear began to discharge foul-smelling green pus. An incision was made

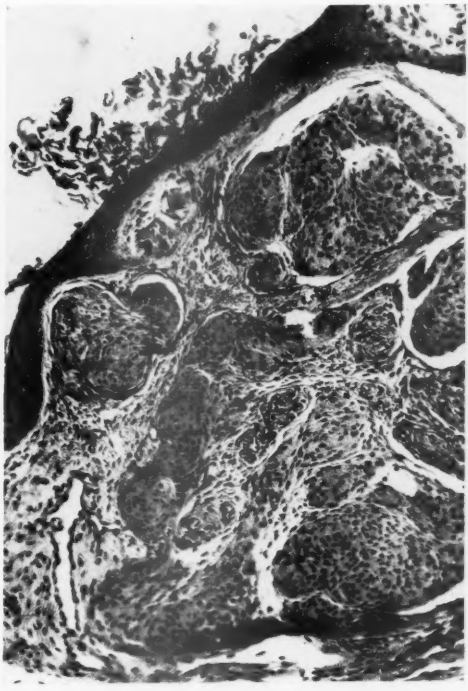

Fig. 4.—Low power magnification of microscopic section of the tumor showing squamous cell carcinoma with only slight hornification.

into the postauricular swelling to release "tension" and since that time she has had drainage from both her external ear canals as well as from a fistula in the upper part of the postauricular incision.

The positive findings on physical examination were as follows: There was considerable malnutrition with evident recent weight loss. The right ear canal was filled with profuse foul-smelling, green, purulent discharge. The lumen was completely obstructed by what appeared to be a polyp or edema of the posterior canal wall (Fig. 1). There was a diffuse swelling over the right mastoid process, extending from the mastoid tip up to the suprameatal crest. A fistula 1.0 cm. long by 0.5 cm. wide was present in the upper portion of the mastoid area. Green foul-smelling pus was observed in the fistula. There was complete right peripheral facial paralysis (Fig. 2) and

an unsteady gait. X-ray studies revealed a destruction of the right petrosa but the semicircular canals seemed intact (Fig. 3).

She was prepared for surgery with blood plasma, a high caloric diet and vitamins.

A large cavity was effected during the operation. It was intended that radium be implanted into this pocket. This procedure was abandoned since the patient developed hoarseness and dysphagia five days postoperatively, due to a paralysis of the right vocal cord and soft palate. The jugular bulb invasion progressed rapidly and in a short time her tongue deviated to the diseased side and examination revealed a loss in power of the right sternocleidomastoid muscle. All further plans for treatment were discontinued forthwith. She expired at home exactly one month after treatment was begun. No postmortem findings were obtained.

SUMMARY AND CONCLUSIONS

1. Primary carcinoma of the middle ear and mastoid process is rare.

2. In most instances, when the patient first comes under observation, it is difficult to determine accurately whether the lesion arises medial or lateral to the tympanic membrane. However, in any case in which there is a marked invasion of the temporal bone with only a correspondingly minor degree of external auditory canal infiltration, it is our opinion that it could be considered a true primary carcinoma of the middle ear and mastoid process. All of our six cases seemed to fall into this category.

3. Long-standing otorrhea and aural pain out of all proportion to the clinical findings seem to be the symptoms most common to neoplasms involving the middle ear and mastoid process.

4. Early diagnosis, combined with complete surgery and radium therapy, seems to be the keynote to more successful treatment.

5. Six more cases of neoplasms involving the middle ear and mastoid process are outlined for addition to the literature, one case being reviewed in greater detail.

1853 West Polk Street.

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LX

DEEP NECK INFECTIONS

Part II

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SUBMAXILLARY REGION INFECTION

By submaxillary region infections are meant those in which the infection first involves the region below the mandible in the submaxillary and submental triangles, the parotid region, and the upper part of the cervical region adjacent to these triangles.

Ludwig's angina comes under this classification. I believe that it is only one of the varieties of submaxillary region infection and has rather definite characteristics. Submaxillary infections may or may not originate in the dental region and may be grouped on that basis.

Submaxillary Infection of Dental Origin. In the majority of instances, infection which gets beyond the teeth and alveolar process becomes a neck infection of the type of Ludwig's angina[24] and as such involves the submental and submaxillary triangles and the upper part of the cervical region adjacent to these triangles. Most frequently it occurs after a tooth extraction though the trauma of the extraction may merely give it impetus, the infection having been already present. The floor of the mouth and the tongue are involved prior to the neck involvement. It originates in the teeth and the alveolar process of the mandible. Most cases of Ludwig's angina are of dental origin, but sometimes the cause is not dental, the source being found in the floor of the mouth, salivary apparatus, base of the tongue or lingual tonsil, or perhaps in the tonsillar fossae, especially when there are tonsil remnants. When there is a lymphatic jump directly into the cervical glands, the typical Ludwig's angina does not occur but a very different condition of localized lymph node swelling takes place, which may or may not go on to abscess formation.

Typical Ludwig's angina presents a clinical history about as follows: Following a dental extraction, the socket does not become

comfortable as in the normal case, but there ensues a distinct discomfort from an inflammatory reaction. This discomfort increases and, in from one to four or more days, becomes severely painful, with swelling and tenderness of the gum. As the inflammatory swelling and pain increase, there is involvement of the floor of the mouth, spreading to the tongue which is pushed upward and backward, and simultaneously there develops a gradually increasing trismus.

At this time it is still possible to visualize the fauces and the oropharynx; also at this time the inflammatory involvement is still entirely above the mylohyoid muscle, that is, entirely oral. Digital pressure upward and backward on the submental triangle, below the chin, is decidedly painful in the mouth, though there is not yet swelling of the neck. As soon as there is involvement of the base of the tongue, any functional movement of the hyoid bone or passive movement by digital palpation is painful. Mastication and swallowing also become difficult and painful. If, at this stage, there is localization and the abscess or abscesses can be drained through the alveolar process or mouth floor, recovery will often take place without involvement of the neck by further extension. If, however, termination of the infection does not take place at such a time, the infection penetrates the mylohyoid muscle diaphragm and invades the neck. This takes place rapidly, and all symptoms and signs are now greatly intensified. The oral involvement is greatly increased, so that it is no longer possible to visualize the fauces and the pharynx because of the extreme trismus. This may be so extreme that the patient cannot separate the incisor teeth more than one-eighth of an inch, if at all. The tongue is greatly swollen and crowded upward and backward against the palate, and on its sides can be seen indentations of the contacting teeth from pressure. Chewing is impossible. This and the trismus are regarded as due to involvement of the internal pterygoid and the muscles attached to the hyoid bone. Salivary and mucoid secretions accumulate in the pharynx because of the inability to swallow. Even breathing may be difficult because of the displacement of the tongue into the pharynx. The whole mouth floor is greatly swollen, the edema rising up to and beyond the level of the free borders of the teeth. There is almost always a foul odor to the breath.

A lateral neck roentgenogram will now show the shadows of the tongue base and epiglottis in contact. The neck in the submental and submaxillary triangles and the adjacent upper cervical region will now be greatly swollen and extremely hard. It is an intense cellulitis, the appearance of which is decidedly characteristic

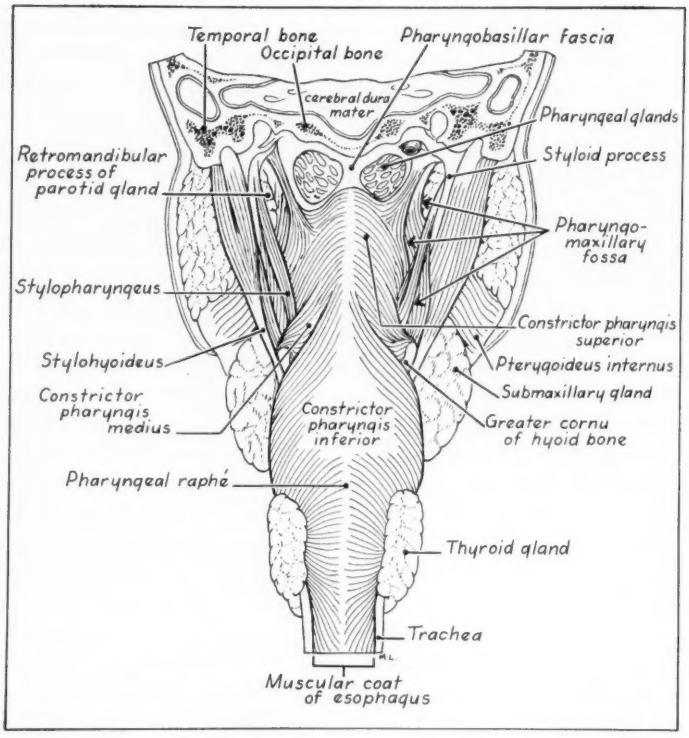

Fig. 13.—Drawing showing the pharyngomaxillary fossa from behind. (After Sobotta, McMurrich, Atlas of Human Anatomy, used with permission of the publishers, Stechert-Hafner, Inc.)

of Ludwig's angina. In fact, unless this cellulitis is present, it is not a typical case of Ludwig's angina. No lymph nodes can be felt in the area. It has almost a bony feel. At this stage edema of the larynx is likely to occur, announced by hoarseness and dyspnea, which may shortly go on to asphyxia if not relieved. When it is severe, tracheotomy will be necessary to restore the airway. McCaskey[25] has suggested severing the fibers of the mylohyoid and geniohyoid muscles for the purpose of relieving the pull on the hyoid bone and diminishing the crowding of the tongue into the pharynx, all at the time of the drainage operation. Orton[26] in addition to doing this also severs

the anterior belly of the digastric. It is possible that, by so doing, one may occasionally avoid tracheotomy. When in doubt the patient will be safer with preliminary tracheotomy.

While all these local manifestations are taking place, the patient is becoming very ill. Fever develops as soon as the oral soft parts are involved. In my experience, the temperature in these cases has been between 101° and 106° F., and at first continuous or sustained. Often, as soon as the infection reaches the neck, there occurs a sharp chill and a sudden elevation of temperature, which indicate that a severe sepsis has developed. The pulse rate is increased. There is apt to be sweating, both day and night. Sweating nearly always follows a chill, but may occur without chills. If more than one definite chill, with a sudden rise of temperature, occurs, it may be concluded that a blood stream infection impends or is present. One chill does not necessarily indicate blood stream infection but it should be regarded with strong suspicion. Daily abrupt rises and falls in temperature indicate blood stream infection, especially if accompanied by chills.

With the disinclination and, later, the inability of the patient to swallow, dehydration occurs, and unless fluids are supplied by subcutaneous or intravenous administration, the condition becomes alarming. There is an increased loss of fluids and of salt from the profuse sweating, and these necessities must be supplied in requisite quantities. The blood count will disclose the usual picture of infection, with leucocytosis. There will be more or less anemia, depending on the duration of the illness; it will be greater in the presence of the hemolytic type of organisms. Transfusions are strongly indicated. The fluid intake should be three to eight liters per day for an adult, the deficit being supplied in the form of five per cent glucose in physiological sodium chloride solution. As soon as a patient develops a definite severe sepsis, drainage is usually indicated. Delay is dangerous.

In these cases of dental origin one must be on the lookout for osteomyelitis of the mandible.[25, 27] If, one or two weeks after neck drainage has been established, there does not occur marked subsidence of pain, tenderness, and swelling in the region of the mandible, osteomyelitis should be suspected. Immediate roentgenograms of the mandible should be obtained, not for the purpose of demonstrating sequestra but for comparison with future roentgenograms.

Roentgenograms taken at this early stage will not show sequestration, but they may show osteoporosis in areas where later roent-

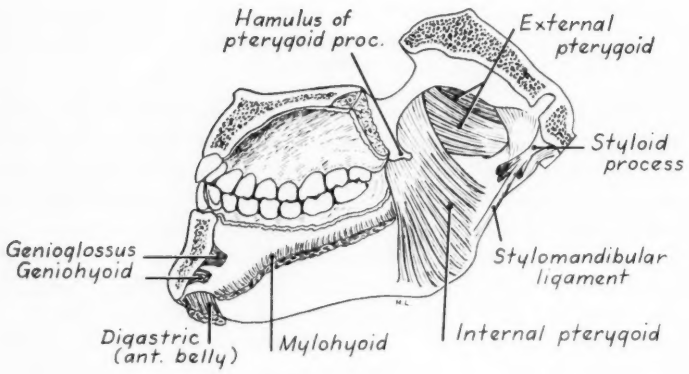

Fig. 14.—Drawing showing the stylomandibular ligament and the internal pterygoid muscle. It also shows the cut attachment of the mylohyoid muscle, which muscle separates the mouth cavity and the neck. (After Sobotta, McMurrich, Atlas of Human Anatomy, used with permission of the publishers, Stechert-Hafner, Inc.)

genograms will show sequestration. In my experience, osteoporosis was disclosed during the second week and osteomyelitis with sequestrum formation two weeks later.

The problem of which teeth should be removed, and when, must always be solved in these cases. It has been our custom to remove any tooth the roots of which are in intimate relationship with frankly suppurating areas. The local drainage will be much improved after such teeth are removed. Even if it is possible without undue risk to preserve such a tooth, there is strong possibility that it will emerge from the disturbance devitalized. In my opinion, the risk of not removing it is greater than the possible gain. This is particularly true in osteomyelitis with loss of bone by sequestration. The aim should be free drainage at all points. Once osteomyelitis with extensive sequestration exists, mastication is to be avoided because of the possibility of fracture of the mandible. At this stage of the illness, the inflammation in all localities except the dental region and the mandible has almost or wholly disappeared. Drainage of the involved part of the mandible should be maintained until the last sequestrum has come away or been removed surgically, after which healing will be so rapid that drainage may be discontinued. I believe that the stage of sequestration should be treated conservatively, waiting until

the sequestra have been completely loosened from the vital bone. Careful exploration with the probe gives valuable information as to when this has occurred. It is not always necessary to make new and extensive incisions for their removal. In some instances the drainage opening already existing will suffice, though it may be necessary to enlarge it somewhat in accordance with the size and location of the sequestrum. It is amazing how quickly the wounds heal after the last sequestrum has been removed. In the surgical management of osteomyelitis of the mandible, the nutrient vessels of the bone should receive the utmost consideration and respect and injuring them should be scrupulously avoided.

Sometimes, when teeth have been extracted from both the upper and lower jaws at the same sitting and when mandibular nerve injection has been performed, it is difficult to determine through which of these portals the infection has entered. It may have entered through all of them simultaneously and the resultant possible pathways of travel and site of localization must be carefully considered and evaluated.

The foregoing description applies when the patient has not received chemotherapy. When, however, one of these drugs has been administered in an early stage of the infection the course will usually be greatly modified. The toxemia, with its attendant general manifestations, will be altered to the extent that the fever and chills will probably not be much in evidence, if at all. If such is the case and the local manifestations become mild, surgical indications are not frankly manifest. If, with such subsidence, progressive improvement to normal takes place, further administration of the drug may be stopped. If, however, after cessation of medication, there is a recurrence of local, and perhaps general, manifestations, it is to be expected that frank symptoms and signs of localization will be present to guide drainage surgery. At such a time, it is my belief that an operation for external drainage should be done, followed with chemotherapy.

It is to be noted that in the typical case of Ludwig's angina there is present a very foul condition in the mouth. In many of these cases it is possible to demonstrate the presence of the fusospirochete organisms of Vincent's angina on smear. Other anaerobic organisms may also be present. For this reason careful mouth hygiene with application of oxidizing agents is an important endeavor. Zinc peroxide, sodium perborate, chlorazene, methylene blue and salvarsan powder, 25%, with boric acid powder are various agents that may

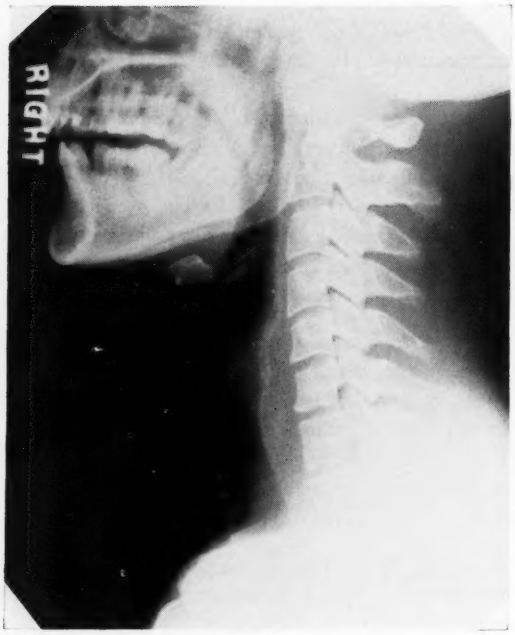

Fig. 15.—Lateral neck radiograph showing normal shadow of the retropharyngeal soft parts and shadow of the normal space between the base of the tongue and the epiglottis.

be useful locally. Hot sodium chloride mouth irrigations are indicated. Administration of neoarsphenamine intravenously, 0.3 to 0.6 grams early in the course of the illness is a valuable therapeutic measure. Mafarsen may be used instead. Alden[1] has reported favorably on this form of medication, stating that it has served well in preventing osteomyelitis.

The application of heat directly to the inflamed parts is valuable and beneficial. Irrigation of the mouth and throat with hot sodium chloride solution having a bag temperature of 110° to 115° F. may be done at two hour intervals. If these are too exhausting for a given patient, the frequency may have to be diminished. External heat or cold may be preferred, and sometimes it is well to be guided by the preference of the patient after trial. External applica-

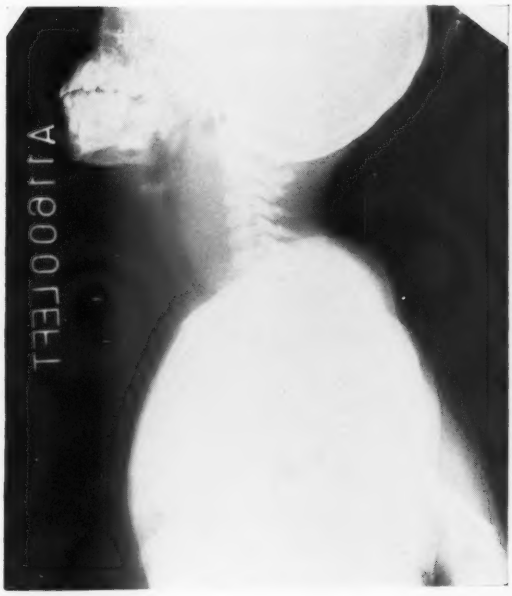

Fig. 16.—Lateral neck radiograph showing widening of shadow of retropharyngeal soft parts when there is present a retropharyngeal abscess.

tion of cold may be used in conjunction with the hot sodium chloride throat irrigations. Irradiation with roentgen ray is sometimes beneficial as auxiliary treatment, particularly when the cellulitis is pronounced. Irradiation should not be used as a substitute for drainage.

Infection in the Maxillary Region. While on the subject of infections of dental origin, a short description of infections beginning in the dental region of the maxilla is considered appropriate, though these do not usually cause infection in the submaxillary region.

Infection in the maxillary region presents a condition which differs from infection originating in the mandible. As in the case of the mandibular type, it most frequently follows extraction of teeth under local anesthesia. Instead of the normally rapid, painless healing within several days, there develops a persistent discomfort, soon to be followed by pain and swelling involving the gingival tissues and, in a few days, extending into the soft parts of the cheek. There

Fig. 17.—Lateral neck radiograph showing the increased size of shadows of the submaxillary soft parts and the tongue, with obliteration of the space between the tongue base and the epiglottis. These changes are found in cases of Ludwig's angina.

is then some elevation of temperature. Sometimes there will be recovery; sometimes not, but instead, a progressive involvement of the whole side of the face and head, including the external ear, the eyelids, nose, and temporal region, and extending downward to and beyond the inferior margin of the jaw into the neck. Simultaneously, the infection travels backward behind the maxilla to the sphenomaxillary, zygomatic and temporal fossae, and upward to the region of the apex of the orbit and sphenomaxillary fissure.[28] Before such extensive involvement has occurred, there has developed a well-marked sepsis, and the patient is very miserable and ill. The eye fundi should be constantly observed in the early stage before it is too late to separate the eyelids. Vision should also be carefully checked. Neuritis of the optic nerve will result in more or less loss of vision from subsequent optic nerve atrophy. Interference

with the action of the extraocular muscles, or fixation of the eye, is also a possibility. The patient may then complain of diplopia, but it should be remembered that the patient will not complain of double vision when the lids of one eye are swollen completely shut. Orbital abscess is another possibility, though I have never observed it from this cause. As the infection progresses, there may be extensive sloughing or necrosis deep in the anatomic fossae mentioned, especially in the temporal fossa. In reaching such an abscess for drainage, it is necessary to penetrate beneath the temporal muscle. An approach through the temporal muscle will reach not only an abscess in the temporal fossa but also more deeply in the zygomatic and sphenomaxillary fossae.

In one of my cases the pathological progression took place so rapidly that when drainage through this approach was finally decided upon, it was considered advisable and necessary to first ligate the external carotid artery in the neck below the area of involvement in order to avoid probable severe hemorrhage in the necrosed tissues. Leisurely and unharassed exposure of the abscess cavity can then be made without hemorrhage. Ligation of vessels in the necrosed tissues may be an extremely difficult or an impossible procedure, to say nothing of the risks involved. A comparatively bloodless field is to be highly desired. If the pharyngomaxillary fossa is also involved, as indicated by the evidence in the throat, this must also be drained. The best approach is the submaxillary, as described by Mosher.[7] If there is a blood stream infection, because of which an internal jugular vein ligation must be done, this fossa may at the same time be drained at the upper end of the same incision. Obviously, when these deep areas are the seat of severe infection, one must consider the possibility of thrombosis of the pterygoid plexus of veins. If this occurs, thrombosis of the cavernous sinus is a possibility, in which case the outcome may be fatal. In the presence of these severe suppurative infections arteries may become eroded or necrosed, with resultant hemorrhage, the severity of which depends upon the size of the vessel involved. In the case of veins, there may also occur erosion or necrosis of the walls, but there is usually no hemorrhage because there is simultaneous thrombus formation. Hemorrhage from the temporal or internal maxillary arteries or their branches requires ligation of the external carotid artery. Meningitis may follow this type of infection.

Osteomyelitis may occur. It should be looked for in the maxilla, in the region of the primary dental infection, and in the bones forming the zygomatic and temporal fossae of the skull and in the walls

of the orbit. It is usually not the severe, fulminating, spreading type that occurs in the bones of the cranial vault, but is more or less localized in the alveolar process at the point of dental infection and in the walls of the maxillary sinus. There will then also be infection of the mucosal lining of the sinus. A persistent foul odor after subsidence of the infection in the soft parts usually indicates infection in the sinus. Operative treatment of such a sinus infection should be withheld until the infection of the soft parts and the sepsis have been controlled. Teeth in relation to frankly suppurating areas of bone should be removed. Because of the proximity of the infection to the pterygoid muscles and to the temporomandibular joint, trismus is apt to be pronounced. It is also apt to persist in diminishing degrees for a long time after the complete subsidence of the infection—weeks or months. Exercises and spring-stretching of the bite are indicated as after-treatment.

Submaxillary Infection of Nondental Origin. In this group there occur some of the severest cases of neck infection. They are placed in this group because the first manifestation of infection of the neck is swelling in the submaxillary and submental regions, which rapidly spreads to the parotid and adjacent upper cervical regions, usually on one side. Often the antecedent sore throat is comparatively mild in degree or has disappeared and objective findings are difficult to perceive. It is characterized by absence of swelling of the pharynx and fauces and usually absence of trismus. In the early stage there are no findings which indicate infection in the pharyngomaxillary fossa. A patient may retire to bed in the evening with a mild sore throat which, during the night, increases rapidly in severity, so that by morning there is swelling involving the submaxillary region. At the same time there is moderate fever. Later the fever may reach high levels. There is usually no trismus; nor is it characterized by foul breath odor. There is some tenderness, chiefly over the infected lymph glands. In from one to three or four days there may occur a sharp chill and high temperature. Infected tonsil remnants in the lower part of the tonsil fossae near the tongue should be searched for and regarded with suspicion. There may or may not be palpable lymph node swelling. Sepsis develops rapidly unless the infection undergoes spontaneous resolution or is controlled by chemotherapy. Tonsillitis, pharyngitis, infected tonsil remnants, sinusitis, and lingual tonsil infection are, one or more of them, the common starting points.

If uninfluenced by treatment, the infection spreads rapidly and the sepsis becomes severe. The pathway of extension or travel is

in the direction of the lymph channels to the nodes on the carotid sheath. The neck swelling may not be extreme; in fact it may actually become less in degree, while the infection spreads and involves the blood stream. Decrease in the degree of swelling is of itself no indication of improvement. The infection may also spread deeply to involve the pharyngomaxillary space, particularly if it has first involved the parotid fascia. It has a tendency to involve other fascial layers and compartments, particularly the carotid sheath. It may be necessary, because of the early threat to the blood stream, to place a drain along the carotid sheath before there is any abscess formation. If this is not done thrombosis of the internal jugular vein may occur early. If the symptoms suggest blood stream infection the vein should be ligated. There is usually no free pus present. I have seen thrombosis with complete disintegration of the walls of the vein and without abscess formation, when drainage has been delayed to the twelfth day of the illness.[22] One must be guided more by the degree of sepsis than by local signs.

These are dangerous and insidious cases. Drainage aims at the sheath and at any area secondarily involved, such as the pharyngomaxillary space or the buccopharyngeal fascia. Early ligation of the internal jugular vein is indicated if there is severe sepsis. Daily blood counts and frequent blood culture examinations should be done. In the presence of severe sepsis and evidence of blood stream infection, neck drainage with jugular ligation should not be delayed merely because of meager local evidence in the neck. One must be careful not to permit the appearance of metastatic abscesses in distant parts to lead one to the conclusion that the infection in the neck is of little importance. Such a patient becomes progressively worse until the original infection in the neck is drained and the internal jugular vein ligated or resected. I have seen this occur while marked reduction in the amount of neck swelling was taking place. Extensive drainage of secondary involvements, with complete disregard of the primary involvement in the neck, will not cure a patient. It is analogous to the management of malignant tumors and their metastases. The primary lesion must not be overlooked.

The effect of chemotherapy introduces a factor which may cause considerable confusion. The beneficial effect of the drug is manifested by a reduction in the severity of the sepsis and the blood cultures are apt to be negative. At the same time there may be no improvement at the point of infection in the neck. In such cases, when it is evident that the process is not being improved as a whole, surgical drainage with continuance of the administration of the drug

is indicated. The optimum time for drainage is not so easily determined while the patient is receiving chemotherapy. There is no other type of neck infection in which involvement of the blood stream may take place so early and so suddenly as in these submaxillary cases of nondental origin. If these cases are not recognized and treated with prompt surgical drainage, the following quotation from Mosher[16] is available for the epitaph on the tombstone: " . . . thrombosis of the internal jugular vein from tonsil infection takes its weekly toll under the guise of septicemia of unknown origin."

VISCERAL FASCIA INFECTION.

(BUCCOPHARYNGEAL AND PRETRACHEAL)

The visceral fascia is regarded as one continuous or uninterrupted sheet of fascia reaching from the skull base to the chest; it envelops the viscera of the neck, namely, the pharynx, the esophagus, the larynx, the trachea, and the thyroid gland. Infection high up near the skull base is designated pharyngomaxillary fossa infection because it involves that space. It also, however, involves the upper part of the buccopharyngeal fascia because the superior constrictor muscle forms the inner wall of the pharyngomaxillary fossa. Therefore by visceral, buccopharyngeal, or pretracheal fascia infections are meant infections which involve this fascia below the level of the pharyngomaxillary fossa or, in other words, below the level of the superior constrictor muscle.

Visceral fascia infection may be primary or secondary. It is primary when the tissues along this layer of fascia are the first to be involved when inflammation extends into the neck. It is secondary when the infection has extended to it from other regions or fascial planes, as for example, extension from the prevertebral fascia or the pharyngomaxillary fossa. Trauma from food, from foreign bodies, and from instrumentation are examples of causes of primary involvement. Late extension from a focus of suppuration outside the tonsil is an example of secondary involvement, as it is at first a pharyngomaxillary or parapharyngeal infection. Infection most commonly involves this layer by penetration from within through the walls of the viscera covered by the fascia. Infection may very readily find an avenue of travel along this fascia, and because it is continuous into the chest, it may involve the anterior or posterior mediastinum, or both. Edema of the larynx is one of its most serious manifestations.

Infections involving the pretracheal fascia are always very serious, especially those following esophageal trauma. Perforation of the esophageal wall by foreign bodies or instrumentation gives rise to the most serious and dangerous type of infection of the neck, for in the majority of instances it almost immediately involves the mediastinum, even before there is any visible evidence of it in the neck. All this may take place in two to four days. It occurs before any protective barrier can be set up by nature. Likewise edema of the larynx frequently occurs before there is definite localization of the neck infection.

Wounds and injuries of the neck may cause infection along this enveloping fascia and infection of the lining mucosa and submucosa of the viscera at the same time. There may be compound lacerations of the larynx, trachea, or esophagus, or there may be lacerations or tears of the mucosa from fracture of the cartilaginous structures of the air passages without any wound of the exterior of the neck. The latter condition occurs after blows on the neck such as are sustained in automobile accidents when the neck is driven forward against the steering wheel or some other part of the car. Edematous inflammatory swelling of the lining mucosa of the larynx, trachea, or even the bronchi with consequent obstruction to breathing has been observed. Bronchopneumonia may become a complication in such a condition. Emphysema is an expected complication in these compound injuries.

The emphysema which follows infection from the esophagus may be caused by gas-producing organisms and is known as infectious emphysema. Air or gas in the tissues may be revealed on roentgenograms. With widespread emphysema there is present a characteristic crepitation on palpation. Changes in the character of the voice, such as dysphonia or aphonia, will occur after injuries involving the larynx. Dyspnea of severe character, and emphysema may be expected. On the other hand, clean-cut perforating bullet wounds which are not accompanied by hemorrhage may disturb the larynx very little if they occur above the glottis.

Infection of the neck coincidental with suppuration in the chest, mediastinal or pleural, has been reported.[29] In such cases there may be little or no evidence in the neck but dyspnea may be a prominent symptom. It has happened that the finding of pus along the viscera was a surprise encountered during the performance of a tracheotomy. In such cases there may be a reservoir of pus, of large dimensions, within the chest. It is entirely possible that the neck infection may

be secondary to the infection in the chest, as when peritracheal and peribronchial lymph node abscesses form. These should be ideal cases for drainage of the mediastinum through the upper opening of the chest cage along the viscera. Abscess in the posterior mediastinum, which is lower in location and does not communicate with the neck, must be drained by the posterior approach through the chest wall after rib resection. Infection along this fascia may be secondary to lymph gland abscess.

It will be obvious that the symptoms and signs of infection along the visceral fascia will be variable and dependent largely on the causative condition. Those developing from a nontraumatic cause will differ from those following trauma in that in the latter the symptoms and signs of the injury itself are also present. Those that result from injury to the air passages differ from those that result from injury to the food passages. Prognosis in cases of pretracheal fascia infection should always be very guarded. Pulmonary complications are common in severe cases. Edema of the larynx is always a possibility. Extensive bronchopneumonia with scattered areas of consolidation and extensive bronchiolitis have been observed at autopsy and under the microscope. Dyspnea, out of all proportion to any demonstrable respiratory obstruction, has been observed. Mediastinal emphysema and pneumothorax may be the reason for this. Tracheotomy is often performed as a desperate effort to prolong life. In many cases it is futile because the dyspnea is pneumonic. When tracheotomy is necessary in a patient with profuse drainage from the fascial plane, pyogenic pneumonia is a possibility because it is then difficult to prevent the pus from getting into the tracheal opening. Such a case, with widespread involvement of both lungs and mediastinum, has been observed.

When a patient whose initial lesion was a peritonsillar abscess develops tenderness and slight swelling of the neck and a dyspneic tendency, it may be assumed that there is extension along the pretracheal fascia, usually lateral to the hypopharynx and larynx. External drainage along the buccopharyngeal fascia and a most intensive course of chemotherapy are urgently necessary in such cases. Sometimes the evidence of infection adjacent to a tonsil is very obscure. Necropsy has, however, verified the evidence in such obscure cases. Sometimes a deliberate search for such a focal point of infection close to the tonsil must be made in the autopsy specimen. Richards[30] has observed such a suppurative tract from the region of the tonsil to the side of the larynx. Roberts[31] reported draining

a perilaryngeal abscess which had extended into the mediastinum through the homolateral pyriform sinus.

The distinctive symptoms and signs of pretracheal fascia infection arise from the viscera involved. Infection lateral to the constrictors of the pharynx immediately causes painful and difficult swallowing. Swelling and redness of the wall of the hypopharynx, at first usually unilateral, should be looked for. The first complaint, when infection originates in the pharynx or tonsils, is sore throat. All these symptoms may at first be mild. One of the first indications that the pretracheal fascia is affected is hoarseness. In infants and children this symptom is not always noticed by the parent and it is soon followed by some degree of dyspnea. In adults dyspnea is apt to be a later symptom. Sometimes there is no actual hoarseness but instead there is a gutteral or muffed voice, indicating that the laryngeal swelling or edema is higher than the vocal cords. A muffled voice may be present with swelling limited to the hypopharynx. It may be possible to elicit tenderness on pressure over the laryngeal and hyoid regions laterally.[32]

There may at first be no visible swelling of the neck. In children there may be preexisting inflamed and swollen glands on one or both sides. Later there occurs some swelling of the neck which, in this type of case, is apt to be moderate. At first it is indurated and diffuse. If localization occurs there may or may not be fluctuation. Occasionally in those in which fluctuation is not elicited, there may be present a pitting on deep pressure with the finger. When present this is a very important sign and may be regarded as evidence of underlying pus. It is my belief that it is more frequently to be elicited than fluctuation. Mirror examination of the larynx and hypopharynx, when possible of performance, reveals involvement of those structures. There may be swelling of the lateral wall of the hypopharynx, the pyriform sinus, and also of the glossoepiglottic fold; also inflammatory swelling and edema of the epiglottis which at first may be limited to one lateral half. If the process extends the swelling then advances to the aryepiglottic fold and the arytenoid region. Still later, the remainder of the vestibule and the ventricular band become edematous, and it is no longer possible to see the vocal cord on that side. Further involvement of the larynx causes dyspnea from involvement of the whole of the laryngeal interior. While this involvement of the larynx is taking place, the infection in the neck is making a corresponding advance and the question of drainage must be given consideration. Body

temperature does not run as high as in the submaxillary cases and chills are not as frequent.

At this stage swallowing is very difficult and there is some danger of fluids getting into the air passages. There may occur regurgitation of fluids through the nose on swallowing. This condition is serious, for the patient ceases to make the necessary effort and dehydration is the result. The fluid intake must be carefully recorded, and if the patient will not or cannot take enough by mouth, it must be given subcutaneously or intravenously. Sometimes a feeding tube may be employed in order to minimize the use of needles through the skin. If the infection involves the esophagus, the tube should be left in place and all food and drink given through it. If the involvement is chiefly laryngeal and tracheal, the patient may much prefer to swallow and accept the accompanying pain. A tube of small caliber is less likely to cause ulceration than a larger one. If there be regurgitation of fluid through the nose when swallowing is attempted, the use of the tube may become obligatory. Feeding tubes left in situ for varying periods of time sometimes cause ulceration of the mucosa and chondritis of the larynx, a most unfortunate eventuality. They should be used only when absolutely necessary. Dehydration must receive daily consideration, and if there is pronounced anemia or hypoprotenemia, fluid administration must be supplemented by whole blood transfusion. In cases in which anemia is mild, plasma administration and amigen may supply such requirements. Specific medications, such as serums, bacteriophages, and antivirus filtrates can be very beneficial when intelligently employed. Transfusions from immunized donors are also valuable. Throat irrigations with hot sodium chloride solution are valuable. Chemotherapy is strongly indicated. Penicillin by inhalation with oxygen should be beneficial for these severe visceral fascia infections.

When there is inflammatory edema of the larynx causing hoarseness and dyspnea, steaming inhalations from the croup kettle and humidifiers are beneficial, and when dyspnea becomes alarming, the oxygen tent, with or without helium, is a life-saving measure until the airway is completely restored by tracheotomy.

Occasionally infection of the pretracheal fascia is caused by infection from the thyroid gland or its fascia. There is a rich distribution of lymphatics at this location and there is a rich blood supply. The chief difficulty in such cases is differential diagnosis which, in the absence of sepsis, is not always easy before there is definite fluctuation. Acute thyroiditis must be ruled out. Higbee[33] has reported

the condition and compiled an excellent bibliography. It should be borne in mind, however, that the infection may be limited to its fascia and the contiguous lymph nodes, and that in such cases there may be no symptoms whatever of embarrassment of the function of the gland itself. It is then purely a cervical fascia infection. After subsidence of the infection the basal metabolism should be carefully checked for possible atrophy with hypofunction of the gland.

The blood picture should be carefully watched. This includes daily blood counts and frequent blood cultures. The carotid sheath is close at hand and a blood stream infection with or without thrombosis is a possibility, though not so common as in other types of neck infection. When laryngeal edema, the most common complication, occurs, the question of scarification arises. If the edema is seen early in the course of the illness and if there is not yet severe sepsis and prostration, it probably does no harm if not done deeply. It does not appeal to me because it does not reach the cause and possibly does open up new avenues of secondary infection.

The following description exemplifies a type of case not infrequently seen in children. A child, from six months to two years of age, gives a history of an upper respiratory infection, sore throat, coryza, tonsillitis, two weeks previously. Frequently, also, there is swelling of the cervical glands on one or both sides. After a few days the fever subsides; the child feels better but still not entirely well. Then there is recurrence of the symptoms and the child is admitted to the hospital. Sometimes the patient has been admitted to the hospital before the onset of dyspnea and sometimes after. The crying voice is somewhat muffled and not clear. The cervical glands are again swollen on one or both sides. Such a recurrent history should arouse suspicion that all is not well in the cervical region. There is a temperature elevation to between 101° and 105° F. and the child refuses food.

General examination is negative except for the usual blood picture of infection. Sometimes examination of the throat of such a child is very difficult and unsatisfactory. Contraction of the muscles of the pharynx, tongue, and palate, the presence of frothy secretions, gagging, vomiting, and struggling even when mummied and held make it necessary to be satisfied with a glance or two in lieu of a prolonged or leisurely inspection. Considerable exhaustion occurs if there is dyspnea. Under such conditions of muscular contracture, moderate swellings of the pharyngeal walls are indeed difficult to perceive and the result of the examination is to leave one in con-

siderable doubt. The tonsils may be large, with or without crypt exudates. If one side alone is involved, that tonsil may appear to be more prominent than the other. That posterior pillar may be thicker than the one on the other side. The posterior pharyngeal wall may not appear to be swollen. Even digital examination may be negative. For such a condition the lateral neck radiograph is indispensable, for it may show indisputable evidence of thickening of the retropharyngeal tissues. This evidence should never be disregarded. The infection in all such cases which I have seen entered through the tonsil. Pharyngeal and parapharyngeal infections take place without definite localization and eventually involve nearly all of the parapharyngeal tissues, even extending around posteriorly to the opposite side.

The question which arises is when and where to drain such a neck in which the place of localization is so obscure. It is useless to incise the posterior pharyngeal wall perorally in such cases. It does not reach the source which is usually in or about the tonsil. When seen before dyspnea is present, I believe that the removal of one or both tonsils, with or without search for an abscess through the tonsil fossa, is rational. If dyspnea is present, I believe that external incision and placing a drain lateral to the pharynx on one or both sides and also placing a drain in the pharyngomaxillary space close to the tonsil bed on the side showing the cervical adenitis, should be done. If pus is found through the tonsil bed, one may delay external drainage until dyspnea appears. If sepsis and dyspnea appear and do not subside promptly, whether or not pus was found through the tonsil fossa, external drainage should be done; if necessary, on both sides, one drain on the sheath and one in the pharyngomaxillary space. When the sheath is exposed it is a simple task to explore the side of the larynx. When there is no actual laryngeal or tracheal obstruction, tracheotomy cannot be expected to relieve the dyspnea. On the contrary, tracheotomy may be harmful. Many have expired with symptoms of asphyxia with a tracheotomy tube in position, Autopsy findings indicate that lung and pleural infection were already existent, and also mediastinitis.

Two autopsies throw considerable light on these obscure conditions. In one patient, a child nine months of age, there was marked inflammatory thickening of all the tissues surrounding the hypopharynx, larynx, and trachea. The airway was free. There was a small abscess at the lower pole of one tonsil, found only after a deliberate search for it. In the lungs there were seen, macroscopically, extensive bronchopneumonia with scattered areas of consolidation;

and microscopically, extensive bronchiolitis. No report was given on the mediastinum. In the other patient, an adult male 56 years of age, the pathologist reported moderate congestion and edema of the lungs, also acute anterior and superior mediastinitis. In the neck there was a diffuse spreading cellulitis involving the muscles, and the entire prevertebral tissue was edematous and reddish-brown in color. An abscess, extending from the tonsillar fossa along the lateral wall of the pharynx to the level of the thyroid cartilage, was found. The pathologist noted that the abscess had been opened by intraoral incision. Microscopic examination showed marked pulmonary edema and infiltration of the parenchyma of the kidneys and liver with leucocytes.

Several deductions suggest themselves from these autopsies. Dyspnea was a prominent symptom in both. In one, the child, there was extensive pneumonia, which was probably the most important cause of death, which was not sudden. In the other, the adult, there was acute anterior and superior mediastinitis and moderate congestion and edema of the lungs, the former probably being the more important cause of death, which was sudden. Both had a small abscess close to the tonsil. I am more and more coming to the belief that some of these deep neck infections may be aborted by timely tonsillectomy and a search for such a focus of pus through the empty tonsil fossa. Sometimes it is possible to cut short a deep neck infection by removal of the tonsils alone. If it fails no particular harm has been done. In some there will be a lead in the form of a granulating tract or a fistula through the muscle bed into the pharyngomaxillary space. In some there will be no lead, and it will be necessary to penetrate the bed with a blunt instrument to evacuate the pus, if there is bulging of the muscle bed. The dissection in such cases is started with a sharp instrument, liberating the pillar margins only, and continued bluntly with a small piece of rolled gauze in the grasp of a clamp, after which the snare is applied. The postoperative healing is practically the same as after uncomplicated tonsillectomy. If a dark blood clot is evacuated it may signify vessel erosion; if there occurs hemorrhage of large or small amounts following operation, it will be necessary to do a carotid ligation. Frequently, following the tonsillectomy, cervical gland swelling immediately subsides and the patient is relieved of the discomfort existing prior to the operation.

What has been said here with regard to tonsillectomy also applies to tonsillectomy when done for the occasional case of inaccessible peritonsillar abscess in the adult. When peritonsillar abscess

forms between the anterior pillar and the tonsil the symptoms and signs are frank and in such cases infection of the neck is rare. These constitute the commonly seen variety of peritonsillar abscess which is frequently opened and drained by the family physician or which ruptures spontaneously. Prompt recovery is the rule. When the peritonsillar abscess forms between the posterior pillar and the tonsil, or lateral to the lower pole of the tonsil, it may not be possible to drain it by peroral incision. In these the symptoms and signs are not frank nor easily seen, particularly if the abscess is small, and infection along the pretracheal fascia may occur. A peritonsillar abscess deeply situated behind, and perhaps below, the tonsil fossa cannot be drained by simple incision through the upper part of the anterior pillar. In any case, deep incision through the lateral and lower part of the anterior pillar is uncertain and removal of the tonsil is safer and more effective. If improvement does not immediately take place external drainage is immediately indicated.

Occasionally trauma from an unmasticated bolus of food or from a fish bone or chicken bone will cause an infection lateral to the hypopharynx. If there is prompt localization, it may sometimes be drained by incision through the laryngeal or esophageal speculum. The head should be low in position and suction simultaneously applied to avoid inspiration of pus into the trachea and lungs. Incision through endoscopic instruments is rational if one is sufficiently skillful in their use. Trauma to the hypopharynx is not nearly so dangerous as trauma to the esophagus. I believe that incision internally through endoscopic instruments should be done only if a prominent localized swelling can be seen.

Summarized, the possible alarming and fatal complications of infection along the pretracheal fascia are: edema of the larynx, pneumonia, pulmonary edema, empyema, mediastinitis, and blood stream infection. The last mentioned is not so common as with infections in the pharyngomaxillary and submaxillary regions.

PREVERTEBRAL FASCIA INFECTION

The characteristic type of infection along the prevertebral fascia is the midline retropharyngeal abscess which occurs most frequently in infants and young children. Such an abscess or purulent accumulation is situated in the areolar tissue of the retropharyngeal space between the buccopharyngeal fascia anteriorly and the prevertebral fascia posteriorly. The infection springs from sources in the nasal chambers, posterior nasal sinuses, adenoid and nasopharynx,

and from there, through connecting lymph channels, reaches the retropharyngeal lymph nodes which are in relation with the bodies of the uppermost cervical vertebrae. As a result of this infection these nodes become the site of abscess formation. When the abscess breaks its bounds in the nodes it spreads and descends and may eventually reach the posterior mediastinum. Infection resulting from a pathological condition of the cervical vertebrae is not at first an infection of this space but may involve it secondarily. In such cases it may travel laterally, pointing into the posterior cervical triangle. High up it would at first be found behind the great vessels in the retrostyloid part of the pharyngomaxillary space. In these the infection is most frequently tuberculous. Infection in the retropharyngeal tissues is a frequent occurrence with visceral fascia infection and has been referred to under that heading. It is then usually a spreading cellulitis without localization and abscess formation and is definitely secondary.

In the typical case of retropharyngeal abscess one of the first symptoms is refusal of the child or infant to take food. At the same time there is a moderate elevation of temperature and the patient gives evidence of discomfort. There may be slight neck rigidity which becomes more pronounced later. As the infection develops and swelling of the posterior pharyngeal tissues appears, there is muffling of the voice, gutteral in character, and later a type of noisy breathing similar to that heard when a hot morsel of food is held in the back of the mouth while awaiting sufficient cooling for swallowing.

If the condition is not seen and recognized at this time and spontaneous rupture does not take place, embarrassment of respiration occurs. The clinical picture then has become grave for several reasons. Usually by this time, because of the refusal to swallow, the patient has developed a marked degree of dehydration, which, with sepsis and embarrassment of respiration, has brought about considerable exhaustion. The first requisite at that time is to relieve the respiratory obstruction. This obstruction is caused by encroachment of the pharyngeal swelling on the laryngeal opening or airway. This swelling quickly disappears if the abscess is incised and drained. If the dyspnea is so severe that any exertion tends to cause asphyxia, a preliminary tracheotomy must first be done. Examination at such a time, visual or digital, is injudicious and contraindicated, and in such cases it should be quite obvious that it may not be possible to arrive at an exact diagnosis until after tracheotomy. If dyspnea is not extreme, a lateral neck radiograph may and should be taken.

If one is sufficiently skillful with endoscopic instruments the abscess may be evacuated through these. There is a possibility of rupturing the abscess when doing this. The ideal time to drain a retropharyngeal abscess is before it has reached the level of the larynx while it can be seen and reached through the mouth. The head should be low and suction at hand.

These midline retropharyngeal abscesses tend to gravitate into the posterior mediastinum along the prevertebral fascia covering the longus colli muscles. Spontaneous rupture sometimes occurs, but an abscess should be opened surgically as soon as it can be diagnosed. I have not seen one which started in the midline take a lateral course, though it is conceivable that it might travel along the fascia of the scalenus anticus. In this case it would reach the carotid sheath which crosses this muscle. It might then extend into the posterior cervical triangle in the lower part of the neck or into the axilla and costocoracoid region.

If peroral incision is made before respiratory embarrassment has occurred, recovery is usually prompt and permanent. External drainage by Dean's[34] approach is useful in those cases in which there is dyspnea, for the evacuation of the abscess would immediately relieve it, and might avoid a tracheotomy. It is sometimes impossible to make a satisfactory examination in such cases, because the patient's struggling while being examined increases the dyspnea. It is unwise to use force. Happily, a lateral neck radiograph gives invaluable aid, as it will always show definite evidence, particularly if the abscess has not already evacuated itself. In those patients who have no dyspnea, digital examination of the pharyngeal wall may be employed to supplement visual examination. Digital examination has not been as helpful to me as I have desired, especially in cases where the visual evidence has been doubtful. I am inclined to place more confidence in the lateral neck radiograph. It should be remembered that the normal width of the radiographic shadow of the retropharyngeal tissues is relatively somewhat wider in the young infant than in children and adults. A widening of the shadow of the tissues between the vertebral bodies and the pharyngeal cavity should never be ignored.

If an infection high up near the skull base is in a lateral position at the start it becomes a parapharyngeal, or pharyngomaxillary, infection, as well a prevertebral. Both the midline and the lateral regions may be involved at the same time. In such a case peroral incision would probably not be conclusive, and if the sepsis persisted, external

drainage of the pharyngomaxillary space would be indicated. Sometimes infection starting from a small abscess in relation with the tonsil and secondary to tonsillitis may involve the retropharyngeal tissues not with abscess formation, but with a diffuse inflammatory thickening which gives evidence of itself in the lateral neck radiograph. Inspection and palpation through the mouth are unsatisfactory in such cases. These patients may have dyspnea without laryngeal edema.

It seems that in cases of retropharyngeal abscess, while the abscess gravitates and heads for the posterior mediastinum from the start, death is more likely to be caused by mechanical obstruction to the larynx or by aspiration pneumonia after rupture of the abscess or by both.

CERVICAL LYMPH GLAND INFECTION

There are two principal types of cervical gland infection. The one involves chiefly the glands of the superficial chain and the other involves the glands of the deep chain. Clinically this distinction may be stressed. The superficial glands lie between the superficial fascia and the superficial layer of the deep fascia, and are therefore in close proximity to the overlying skin. They are particularly numerous along the external jugular vein and its tributaries. They are found under the skin of the entire cervical region and drain into the deep chains. The deep chains are arranged in certain, more or less definite, group chains; some chains horizontal, others vertical or oblique, and others irregularly. They are situated at all fascial depths, that is, all layers of the deep fascia are plentifully studded with lymphatics. Clinically, the deep chains of glands of both the head and the neck should receive combined consideration. In fact, the same group may be located in the head and in the neck, as, for example, the parotid and preauricular lymphatics.

High up in the retropharyngeal space there are several lymph nodes which take drainage from the nose and the nasopharynx. When these suppurate a retropharyngeal abscess is the result. In a more or less horizontal line from the occiput to the chin there are several groups of nodes, namely, the suboccipital, the posterior auricular or mastoid, the preauricular and parotid, the submaxillary, and the submental. In the posterior cervical triangle there are glands in close series with the sheath glands. They extend from below the occipital and the mastoid bones to the clavicle and the scapula. Those in the parotid group are found on the fascia of the gland and are

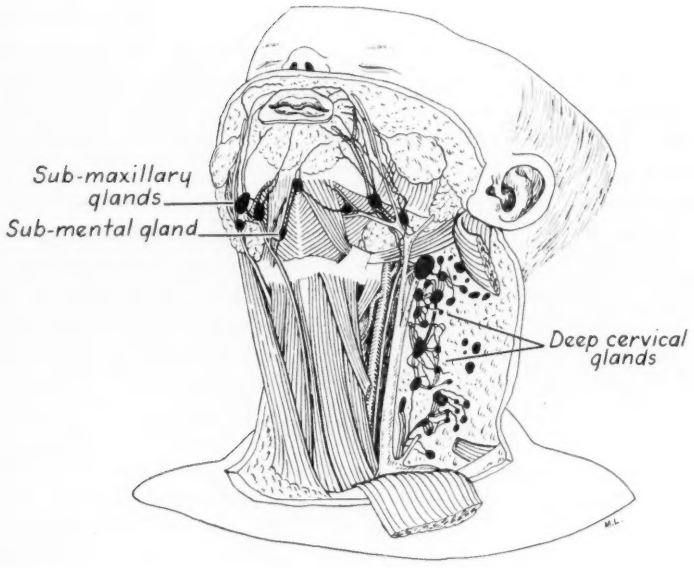

Fig. 18.—Subcutaneous neck lymphatics in new-born child. (Dorrendorff.) (Piersol's Human Anatomy. By permission of the publishers, J. B. Lippincott Co.)

also enmeshed in the parotid gland structure. Swelling over the parotid region, closely resembling mumps, may be caused by inflammatory swelling of these lymph glands alone.

Receiving the drainage from all lymphatics of the head and neck is the large, deep chain situated along the carotid sheath beneath the sternomastoid muscle and arranged in a more or less vertical direction. This is the most important lymphatic group in the neck. When inflamed the nodes become quite adherent to the fascial sheath of the vessels and it may be assumed that blood stream infection frequently occurs in this way.

Inflammation of the Superficial Glands. The sepsis is not so severe and is usually of short duration. There is a greater tendency to abscess formation, and when this occurs it is superficially located, subcutaneous. Fluctuation occurs frequently and is readily elicited.

Inflammation of the skin, manifested by redness, often occurs. In the abscence of severe sepsis and in the absence of involvement of the deep lymphatics, it is justifiable and wise to wait for fluctuation; in fact, it is the only type of neck infection about which that can be stated. There is no involvement of the fascial plane except at the point of suppuration. There may be spontaneous subsidence without suppuration. There may be extension to the deep chain with resultant increase in the severity of the infection. When this occurs the condition should be regarded with all the caution that applies to a fascial plane infection. Even after a superficial abscess has been incised and drained this extension may occur.

There is no justification for the use of the aspirating needle, as fluctuation is recognizable as soon as abscess formation occurs, in which event the scalpel is indicated. Aspiration with a needle may produce a widely spreading cellulitis where none existed previously; when abscess of the deep chain is suspected, exploration with the needle is even more objectionable for the same reason and also because of the greater danger of perforating deeply lying blood vessels. In these, edematous pitting can often be elicited.

Inflammation of the Deep Glands. With inflammation of the deep glands the sepsis is more severe. Suppuration occurs later and does not so often take place. Fluctuation is therefore much less commonly elicited, but pitting on deep pressure with the finger is often present when there is no fluctuation. Often the individual glands cannot be felt and there is only a fullness along the sternomastoid muscle or a diffuse prominence on that side of the neck. There is frequently spontaneous subsidence of such symptoms and signs, with recovery. This is frequently observed by the pediatrist. The condition may remain more or less stationary for several weeks and suddenly change into an alarming status by extension and the development of a most profound sepsis. Blood stream infection with distant complications may occur suddenly and without warning. Constant, close watching is necessary. A severe chill is sometimes the first sign.

When infection strikes into the neck from the portal of entrance in the upper respiratory areas, the lymphatic system bears the brunt of the initial invasion and gives the first visible evidence. In this early period there is usually a sustained elevation of temperature. Later, if the temperature is characterized by abrupt rises and drops, especially if accompanied by chills, the blood stream has been in-

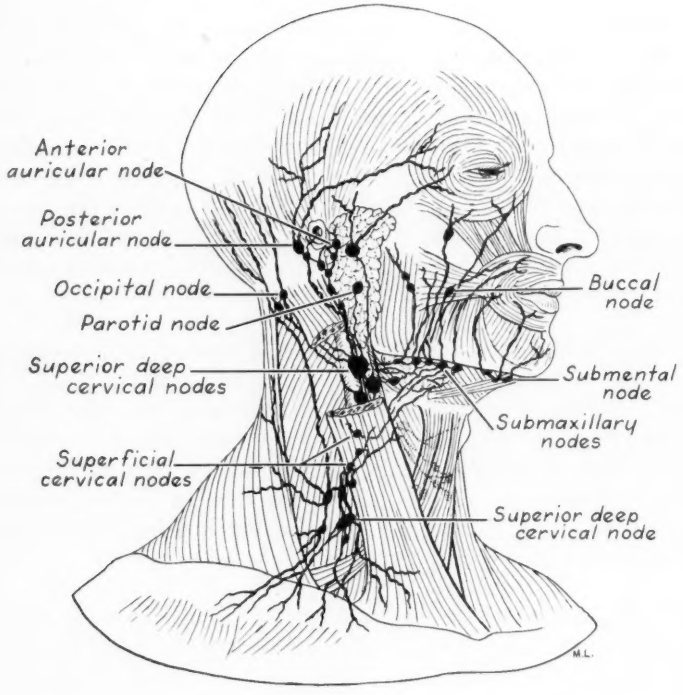

Fig. 19.—Superficial lymphatic vessels and glands of head and neck; semidiagrammatic. (Piersol's Human Anatomy. By permission of the publishers, J. B. Lippincott Co.)

vaded. Sometimes this invasion of the lymphatics of the neck occurs with direct invasion of the tissues of the neck by continuity from the portal of entrance, as, for example, the teeth. In other instances there is a lymphatic jump from the portal of entrance through lymph vessels directly to the lymph nodes of the neck, across fairly large areas of uninvolved tissues. I have observed this to occur from the pharyngeal mucosa and from the buccal side of the alveolar process of the mandible. At that time it constitutes the whole of the neck infection.

Usually when there is improvement in the gland inflammation it is of favorable prognostic significance, other features not indicating

the reverse. Observance of the behavior of the lymphatic inflammation is of the utmost importance. It happens sometimes that the inflammation at the portal of entrance subsides completely, leaving the lymph node inflammation as the only remaining evidence of the infection. This happens frequently after tonsillitis. After a fairly long delay of this nature a deep fascia involvement may still occur. Sometimes deep infection along the fascial planes is not accompanied by visible involvement of the lymphatics. It is in such cases that there is little or no visible swelling of the neck. Infection of the pretracheal or visceral fascia is often present without swelling which can be recognized.

The nodes in the neck that are most frequently involved are those situated at the anterior border of the sternomastoid muscle just below the angle of the jaw. They lie about over the bifurcation of the carotid artery. Infections entering through the pharynx and tonsils very frequently cause inflammatory swelling of the glands below the jaw angle, and that almost immediately. This is a typical lymphatic jump. There is usually high temperature and there may be one sharp chill. Infections in the submaxillary region differ in that this region swells and becomes very much indurated, and the individual glands cannot usually be palpated. With infection along the pretracheal fascia, lymph node swelling is not so common and often none is palpable. Occasionally there is a lymphatic jump from infection in and about the teeth, especially when occurring in children. In such cases the glands may break down and form an abscess. When there has been inflammatory swelling of glands for more than several weeks an abscess frequently results, but there may, on the other hand, occur spontaneous subsidence of the inflammation even in such prolonged cases. Glands which have been swollen for several weeks or more may undergo late suppuration with the formation of an abscess, without the development of high fever. If such an occurrence is capable of being determined by fluctuation or pitting, the abscess should be drained.

An important question to answer when there are inflamed glands is where the infection entered. If the patient is seen early and during the initial attack this evidence will be found somewhere in the upper respiratory tract, teeth or temporal bones. If the patient is seen later the original inflammation in these parts may have disappeared. In evaluating the history a red throat should never be forgotten, never ignored.

The management may be summarized as follows:

1. With involvement of the superficial glands chiefly, one may wait for localization and abscess formation, provided there is no sepsis. As soon as an abscess forms, as manifested by the usual signs, incise and drain, even if there is no sepsis.

2. If no abscess forms and there is no sepsis, wait. In such cases there may occur spontaneous subsidence.

3. If deep glands become involved, wait for signs of abscess formation but be on the alert for sepsis and fascial plane involvement. If sepsis and extension occur, drain. At first, when the inflammation is chiefly lymphatic, there is usually a sustained high temperature. Later, when sepsis intervenes, there will be abrupt rises and drops in temperature and perhaps chills. When this occurs drainage is indicated. A positive blood culture indicates ligation or resection of the internal jugular vein. With chills and sweats and a negative blood culture it is safer to drain.

4. The deep glands may remain enlarged for weeks, and if there is no sepsis, drainage may be postponed. Many of these recover spontaneously.

5. In cases in which severe sepsis and distant complications indicate that a blood stream infection is present and that drainage and jugular ligation must be done, the inflamed, swollen glands on the sheath should be carefully excised, if only to afford proper access to the sheath. In such instances, dividing the belly of the omohyoid muscle greatly facilitates the operative procedure.

It should be remembered that any infection of the head or neck which is the source of infection in the glands can thereby be the cause of a deep neck infection. In the majority of cases the trouble will be found in or near the tonsil and the remedy is obvious. When to remove the tonsils is often a controversial question. In properly selected cases it is possible to drain the neck infection through the tonsil fossa, after which there is usually an immediate and complete subsidence of the gland infection, usually in from two to four days. When there is frank primary acute tonsillitis, tonsillectomy is never to be considered. Iglauer[23] has recommended that when there is neck infection requiring external drainage, this be done, and that the tonsils be removed at the same time. A peritonsillar abscess in child or adult, which has existed longer than the usual intense acute stage of involvement, sometimes becomes encapsulated and lined with granulation tissue. In these there is often a

fistulous communication with the same character of lesion in the neighboring parapharyngeal area and such a condition is cured by tonsillectomy. Any cervical gland inflammation which may have been present immediately subsides unless there is already abscess formation in the glands.

SURGICAL APPROACHES TO INFECTIONS OF THE NECK

Obviously, when an abscess is comparatively superficial and has become subcutaneous, the drainage incision should be made directly into it through the overlying skin at the point of fluctuation. Some abscesses, primarily in the lymph nodes, may be located in any locality of the neck and present no problem of approach. Moderate hemorrhage from veins sometimes occurs and is usually readily controlled with gauze packing. Such an abscess is apt to be lobulated and, if so, all soft-tissue septa or partitions should be gently broken with the finger, making one chamber.

The incision should be large enough to amply expose the entire abscess cavity. Incisions made in a horizontal or encircling direction heal with less scar tissue contracture owing to the encircling direction of the elastic fibers in the skin of the neck. Longitudinal or oblique incisions run across these elastic fibers and tend to exaggerate scarring. However, it should be recognized that surgery for neck infections is not cosmetic and a scarred living patient is preferable to an unscarred dead one. Therefore an incision along the anterior or posterior border of the sternomastoid muscle, though longitudinally oblique, is to be used without regard for cosmetic considerations when it offers the best access to the particular condition existing. This applies particularly to cases in which it is necessary to expose much of the internal jugular vein and when the major part of the chain of deep cervical glands must be exposed. Through such an incision over the anterior border of the sternomastoid muscle one may reach practically all of the layers of the deep cervical fascia.

The skin incision is of minor importance providing that it is made long enough to completely expose the parts. An approach through a short incision in the neck makes it necessary to work in a small, deep, funnel-shaped hole in which it is extremely difficult to see clearly, particularly if there is the slightest bleeding. It is for the same reason, namely, cramped quarters, also difficult to find and control the bleeding points. A large incision with well loosened and well retracted flaps avoids all such difficulties and gives a flat and shallow field. Working in such a field is not only more comfortable

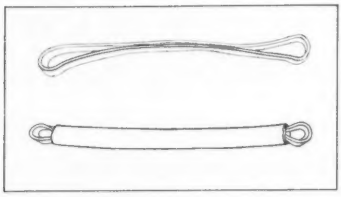

Fig. 20.—A type of soft rubber drainage material devised to avoid extrusion from wounds in parts where movement cannot be entirely eliminated. It does not require suturing or clipping to the wound margins. It is composed of an ordinary rubber band over which is cuffed a piece of Penrose tubing of suitable size as shown in illustration. The free loop at the end allows soft tissues to fall into its confines, thus tending to hold it fixed. It has been quite satisfactory where extrusion of drains must be avoided, and where, otherwise, some type of gauze packing would be necessary. The shorter the cuff, the larger the size or diameter of the loop.

but infinitely safer. Veins are seen before they are accidently injured and may be leisurely divided between two ligatures.

In the neck, bleeding must be under secure ligature control. Pressure may not be applied. In the necrotic pyogenic wall of an abscess cavity, bleeding from small sources may be controlled, if necessary, with gauze packing. As mentioned elsewhere cellulose gauze which is decidedly hemostatic is the preferred material in such conditions. It may be employed as packing around a rubber drainage tube. A soft rubber tube drain is to be preferred if there is no bleeding, because drainage is free along it and it does not act as a cork. Flushing the wound gently with mild antiseptic solutions may be done along such tubing without causing its extrusion. A type of drainage material which has served very well in neck wounds is that shown in Fig. 20. It consists of a rubber band, ¼ to ½ of an inch in width, over which is cuffed a piece of soft rubber tubing of such size as to fit snugly. The cuff of tubing is shorter than the rubber band, thus leaving a loop of band exposed at each end. When this is placed in position in the depth of the wound the soft tissues protrude into the loop of the band, tending to hold it where it is placed and tending to prevent its extrusion from the wound. A probe or grooved director or slender forceps may be used to place the drain.

In localities like the neck where there is more or less movement and which cannot be immobilized, the tendency to extrusion of drains must be prevented. In my experience it has rarely been necessary to clip or suture this drain to the skin margins. The loop drain may be removed a fraction of an inch daily until it is completely out. If the seat of the infection has been reached the choice of drainage material is not of paramount importance provided that the drain is kept in position.

The following is a brief outline of the surgical approaches to the several types of deep neck infection.

1. Prevertebral fascia infection or retropharyngeal abscess.
   a. Incision of the pharyngeal wall directly through the mouth.
   b. Incision along the anterior border of the sternomastoid muscle, as recommended by Dean.
2. Pharyngomaxillary space infection.
   a. Approach through the submaxillary fossa after elevating the gland, as recommended by Mosher.
   b. Approach beneath the angle of the jaw directly.
   c. Approach through the posterior triangle.
3. Submaxillary, submental and parotid space infection.
   a. For submaxillary space infection—incision parallel to and below the inferior border of the jaw, exposing and elevating the submaxillary salivary gland from below if it is desired to reach an abscess above the level of the mylohyoid muscle.
   b. For submental triangle infection—incision, horizontal or vertical, in the submental triangle between the symphysis and the hyoid bone, exposing the deep fascia. Then vertically through the median raphe between the suprahyoid muscles. This may be termed the midline suprahyoid approach.
   c. For parotid space infection—incision over the prominence of the parotid swelling parallel to the fibers of the facial nerve.

4. Carotid sheath and visceral fascia infection.

    a. Incision along the anterior border of the sternomastoid muscle as for jugular resection.

5. Posterior triangle infection.

    a. Incision along or behind the posterior border of the sternomastoid muscle.

Two types of infection may sometimes be drained by internal or peroral incisions, namely, the typical retropharyngeal abscess in infants and young children, and infection localized in the floor of the mouth. The typical retropharyngeal abscess is quite easily opened through the mouth. With suction at hand the head should be placed in position lower than the chest, so that aspiration of pus into the deep air passages is avoided. A mouth-gag should be securely inserted, and a blunt instrument such as the closed blades of nasal scissors or a long clamp quickly thrust through the abscess wall and opened just before removal. All this should be done quickly, especially if there is dyspnea. The gag minimizes swallowing.

In selected cases it is sometimes possible to open an abscess in the floor of the mouth by peroral incision. One must avoid injuring the lingual vessels and the lingual nerve. In the medial and anterior region of the mouth floor all of these structures are situated on the tongue side of Wharton's duct. Posterolaterally the lingual nerve lies on the outer side of the duct, crossing beneath it to reach the tongue. When attempting to reach the deeper layers the blunt instrument is safer than the scalpel. If the abscess is not fairly superficial peroral incision is contraindicated and the external approach should be used. If the abscess is laterally situated the best approach is Mosher's submaxillary route. If it is medial in location the suprahyoid approach through the midline between the muscles may be the best choice. It has been noted that the scar which results after the longitudinal midline incision through the skin in the submental region has a decided tendency to distort the region by excessive contracture. Horizontal skin incisions are not so apt to do this. For the midline approach the skin incisions may be transverse or longitudinal.

Prevertebral Fascia Infection or Retropharyngeal Abscess.—When patients with retropharyngeal abscess, usually infants, also exhibit an extreme dyspnea, nothing can be done, not even an ex-

amination, until the airway is restored. If the dyspnea is so severe that a simple examination of the throat threatens to cause asphyxia, tracheotomy must first be done to completely restore the airway. After that the abscess may be drained leisurely by the internal or external route. If the infection has not reached the mediastinum and is not below the level of the larynx peroral incision should be adequate. If, however, the infection is already in or near the mediastinum external drainage should be done. If the abscess is close to or in the chest cage a drain should be inserted into the upper part of the chest along the esophagus (cervical mediastinotomy[35]). When possible the mediastinum should be drained on the right side because the esophagus is farther away from the dome of the pleura than it is on the left side.

The approach for external drainage of retropharyngeal abscess has been described by Dean.[34] The incision is made along the anterior border of the sternomastoid muscle, exposing the carotid sheath. The muscle and the sheath are retracted laterally. Usually the attack is made above the level of the larynx. One should identify the superior thyroid vessels, retracting them medially. The hypoglossal nerve should also be identified and avoided when working at its level. Below the level of the larynx the infrahyoid muscles, the superior thyroid vessels and the thyroid gland should be retracted medially. The recurrent laryngeal nerve will probably not be visualized, but manipulations should avoid the region close to the trachea and esophagus. The sympathetic nerve behind the carotid sheath should also be avoided. The abscess will most often be found at the level of the hypopharynx. After it is located, a blunt instrument such as a closed clamp is thrust into it, and the opening enlarged by separating the jaws of the clamp. The retropharyngeal space, that is, the areolar tissue region in the midline of the neck between the prevertebral and the buccopharyngeal fasciae, is limited laterally by a union of these two layers of fascia. In reality the union is at or near the deep side of the carotid sheath. This layer, connecting the sheath with the viscera of the neck, is sometimes called the alar fascia and it is by penetration of this layer that access is had to a retropharyngeal abscess.

If it is necessary to drain the mediastinum, cervical mediastinotomy, the incision is carried to the sternum and the trachea and esophagus are exposed. Having exposed the side of the trachea and esophagus, the finger is inserted into the mediastinum along the esophagus and the drain is inserted into the opening thus made. If dyspnea is not severe enough to prevent inspection of the throat and

Fig. 21.—Drawing showing Mosher's T-shaped incision for the Mosher submaxillary fossa approach to the pharyngomaxillary fossa. (Used with permission of Dr. H. P. Mosher.)

there is no danger of asphyxia, this external approach of Dean might make it possible to avoid tracheotomy. General anesthesia of any kind is contraindicated just as in any other condition which is associated with dyspnea.

Pharyngomaxillary Infection. The submaxillary fossa approach as described by Mosher[7] in 1929 is, I believe, the most important procedure pertaining to emergency head and neck surgery since the advent of tracheotomy. If the submaxillary gland is elevated as directed, the operation almost performs itself.

Mosher recommended a liberal T-shaped incision which, after wide elevation and retraction of the flaps, gives a wide shallow field with maximum visibility. The crossbar of the "T" runs parallel with the lower border of the jaw and about one-half inch below it. The vertical portion runs downward just anterior to the anterior margin of the sternomastoid muscle. The incision goes through skin, subcutaneous fat, superficial fascia and platysma muscle down to the superficial layer of the deep fascia which covers the salivary glands. After exposure of the gland the first structure encountered is the facial vein which usually runs across the lateral part of the

submaxillary gland and superficial to it. The vein is divided between two ligatures.

Now comes the most important step of the operation, namely, identifying and elevating the submaxillary gland. Its fascia is sometimes rather dense in the presence of inflammation. It must be identified and incised just above the hyoid bone. If the hyoid bone is pushed into prominence with the finger from the opposite side of the neck, determination of the correct level is greatly facilitated. This having been determined, the fascia is incised, whereupon the gland looms into view. The inferior border of the gland is then elevated with a blunt instrument or finger, working outward toward the lateral part of the gland. At this location one must avoid injuring the facial artery which either enters the gland or courses along beneath it to reach the border of the jaw. When the gland is lifted the artery goes with it. The most difficult part of the operation has now been accomplished. The finger is then inserted under the lateral part of the gland in the direction of the angle of the jaw and the stylomandibular ligament felt. It is then carried upward toward the styloid process deep to the mastoid tip. When the process is felt the finger passes upward beneath it to the base of the skull. It is then in the pharyngomaxillary fossa. It is practically impossible to make a false passage with the finger for this is the path of least resistance and one gets the sensation that there is no other place the finger could go. If there is a pharyngeal fistula the finger might enter the pharynx. In such cases there is no point in enlarging such a fistula and care should be exercised to avoid that. An abnormally long styloid process may be somewhat troublesome to pass; a short one is an advantage. Identifying the stylomandibular ligament is not of great importance, except that it gives one the assurance that the finger is close to the inner surface of the mandible and will reach the styloid process.

The region at the lower pole of the tonsil, where small abscesses are frequently located, is also exposed by this procedure. By coming forward and inward at the proper level the region at the base of the tongue and floor of the mouth may be palpated and explored with the finger. Abscess in the mouth floor is thus reached and evacuated. In such cases there is sometimes a fistula communicating with the mouth cavity. There is no point in enlarging such a fistula, as drainage through the external wound will be adequate.

It cannot be too much emphasized that the most important step in this operation is proper exposure and elevation of the sub-

Fig. 22.—Incision for midline suprahyoid approach in cases of the submaxillary type of infection (Ludwig's angina); through skin and subcutaneous tissues above hyoid bone.

maxillary salivary gland. To walk into a house the door must be opened.

In the submaxillary type of case such as Ludwig's angina in which the tissues are greatly swollen with inflammatory infiltration, recognition and identification of structures and tissue planes are indeed difficult, and one must proceed with courage and caution but with precision. One must identify the hyoid bone and penetrate the deep fascia above its level. On the cadaver it is simple. One must learn to work in these inflamed tissues. There is often as much as two to five cm. of cutaneous and subcutaneous edema, the more so in an obese patient. Blind stabs in the dark are at best extremely dangerous.

At the conclusion of the operative procedure the drain must be carefully inserted into the pharyngomaxillary fossa or wherever the abscess was encountered. As previously stated, I prefer the soft rubber cuffed drain. In cases in which the internal jugular vein has not been exposed for ligation, the vertical part of the incision may be closed with sutures and perhaps also the ends of the horizontal part. In partial closure of any infected wound I prefer chromic catgut skin sutures because they resist infection. Metal skin clips may be used. In those cases in which ligation or resection of the

internal jugular vein has also been done, a soft tube drain should be placed on the sheath at that place and brought out though the lower end of the vertical incision. After three to five days, depending on local and general conditions, all drains are pulled out about one-half inch daily, at the time of dressing, until they are all out. If local and general symptoms and signs indicate that there is still considerable inflammatory swelling and discharge in the pharyngomaxillary fossa, drainage should be continued until all local and general signs of inflammation and sepsis have disappeared. In cases in which there is no necessity for exposing the carotid sheath the vertical part of the incision may be omitted, but in that event the horizontal incision should be a little longer so that adequate exposure may be accomplished by wide retraction of the flaps. From actual experience I have found it advantageous to insert a Carrel-Dakin tube alongside of the drain, long enough so that the outer end protrudes externally though or alongside the dressings. The wound may then be irrigated or flushed every hour or two with penicillin solution in a strength of 250 to 1000 units per cc.

Approach At Angle Of The Jaw. A very rapidly performed and direct approach to the pharyngomaxillary fossa may sometimes be desirable. Such an approach is available by blunt penetration of the deep fascia exactly contiguous to the angle of the jaw. At this point the fascia is tough and dense because it is within the area where the superficial and deep layers of fascia covering the salivary glands come together in union between the submaxillary and parotid salivary glands. If a closed clamp is thrust through and the opening made large enough to permit the introduction of the finger, this may be passed upward to the styloid process and into the pharyngomaxillary fossa. More actual force is required to carry out this maneuver than is necessary by the submaxillary route. It is important that the finger keep close to the internal pterygoid muscle on the inner side of the jaw angle as it is advanced.

Submaxillary Space Infection. In cases of Ludwig's angina type of infection, whether of dental etiology or of nondental etiology, the submaxillary approach is the appoach of choice. Through this access to any part of the region above the mylohyoid muscle may be had. If it is also desirable or necessary to explore the carotid sheath the T-shaped incision gives access.

It has been suggested that in cases of Ludwig's angina with dyspnea from backward displacement of the tongue, such dyspnea may be decreased if the pull on the hyoid bone, caused by the tension

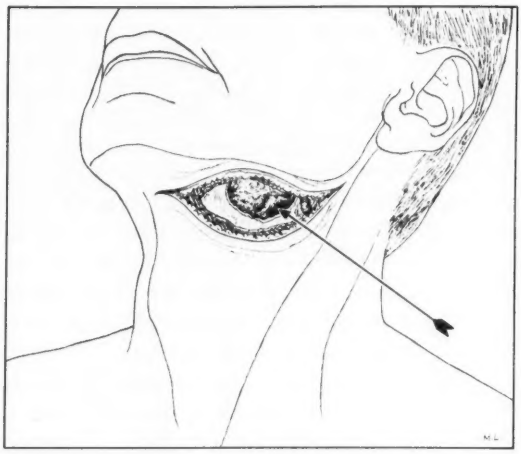

Fig. 23.—Drawing showing surgical exposure of the submaxillary salivary gland. Shaded area (arrow) at the lateral aspect of the gland is the place where a blunt instrument or finger is inserted to reach the pharyngomaxillary fossa.

of the suprahyoid muscles, be released by severing the fibers of some of these muscles. It has been suggested[25, 26] that this may be accomplished by severing the fibers of the mylohyoid and the geniohyoid muscles and the anterior belly of the digastric muscle. In doing this the incisions through the muscles should be placed medial to the hypoglossus muscle to avoid injuring the hypoglossal nerve and the lingual artery and vein.

Submental Space Infection. When the infection is predominant in the midregion of the neck, an approach between the muscles of the two sides of the tongue in the mouth floor, in the midline, may be the most direct. The incision through the skin may be horizontal or vertical. The deep fascia is exposed and incised in the midline from the symphysis to the hyoid bone, and the approach is continued through the median raphe between the muscles of the two sides. The tongue may thus be explored throughout its entire thickness, and at the same time the finger or a blunt instrument may be used to explore laterally to either side at the several levels of muscle planes. No vessels or nerves are encountered.

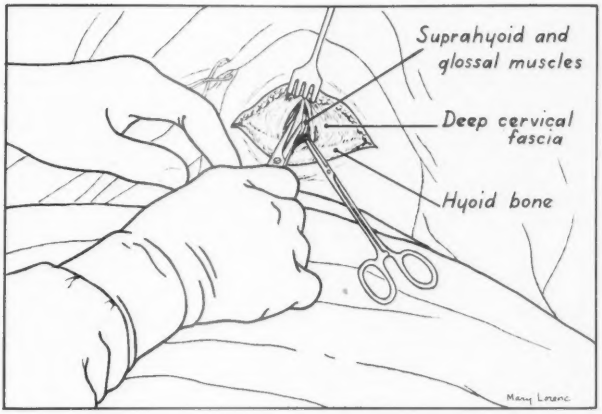

Fig. 24.—Insertion of closed forceps through midline incision of cervical fascia, into median raphe of mylohyoid and lingual muscles. This midline incision through the deep fascia extends from symphysis menti to hyoid bone.

Parotid Space Infection. For abscess in the parotid fossa the incision should be made parallel with the branches of the facial nerve to avoid severing them. The skin incision may be single but the capsule of the gland may be opened in several places. When the infection first involves the retromandibular or pharyngeal process of the gland, from the parapharyngeal area, it is best dealt with by draining the pharyngomaxillary fossa through the submaxillary approach. For parotid abscess Furstenberg[36] advocates a vertical incision through the skin just anterior to and close to the auricle, running from the zygoma downward and curving backward just below the lobule of the ear to the mastoid tip, thus exposing the fascia of the parotid gland. The fascia covering the parotid gland is rather firmly connected to the overlying skin. Then through horizontal incisions through the capsule of the gland the abscess or abscesses are drained. It is important not to sever the facial nerve in making the approach through this incision.

Bezold's abscess may be drained by incising over its prominence just below the auricle and directing the approach toward the digastric groove and root of the styloid process, beneath the sterno-

mastoid muscle from its anterior border. By using the finger from below the parotid gland, deep extension inward may be followed and reached. At the same time a complete simple mastoidectomy should be performed if it has not already been done. By performing the mastoidectomy first, the abscess will be reached when the tip is removed. The incision through the soft parts may then be extended downward and forward to increase the exposure.

Visceral Fascia Infection. Incise over the abscess if one has formed. In those in which there has been no localization the incision is made along the anterior border of the sternomastoid muscle, exposing and identifying the sheath. The muscle and the carotid sheath are retracted laterally. Then the larynx, trachea, and esophagus are exposed, identified, and retracted medially. The drain is placed along these viscera.

Posterior Triangle Infection. Incise over the abscess if one has formed. If none has formed incise over the posterior border of the sternomastoid muscle. In making the exposure the spinal accessory nerve, the branches of the cervical plexus at the posterior border of the muscle, and the transverse and deep cervical arteries are to be avoided. The external jugular vein may require ligation. In the supraclavicular region the deep arteries and veins of the arm must be avoided. In the upper part of this triangle an approach to the deeper parapharyngeal region may be made along the scaleni muscles and beneath the carotid sheath, being careful not to injure the cervical sympathetic nerve.

Occasionally there occurs a type of pharyngomaxillary infection which extends into and involves the deep glands in the upper part of the occipital triangle and under the upper part of the sternomastoid muscle. There is swelling of the lateral pharyngeal wall behind the tonsil but there is no trismus. The infection is in the posterior part of the phayngomaxillary space. There is often abscess formation in the glands and fluctuation is then elicited. In such cases the most direct approach is through an incision along the upper part of the posterior border of the sternomastoid muscle. When the deep glands and the deep fascia have been exposed the finger may be passed upward and inward along the prevertebral muscles to the pharyngomaxillary fossa. If there is present abscess formation the pus will be encountered as soon as the deep fascia is exposed, and the finger passes to the deepest part of the abscess cavity without resistance. This may be termed the posterior triangle approach.

ANESTHESIA

Anesthesia is an important problem in the surgical management of deep neck infection. First of all, the anesthetist must be thoroughly familiar with the difficulties which the situation involves and must see to it that a free airway is at all times maintained. It is obvious that when there is dyspnea, general anesthesia is out of consideration until the airway has been completely restored. When the encroachment is limited to the tongue and pharyngeal region and when this can be completely corrected by the insertion of a pharyngeal breathing tube or possibly by an intratracheal catheter introduced through the nose, the use of general anesthesia is safe. When dyspnea is caused by edema of the larynx or by a pathological condition in the deep respiratory tract, a general anesthetic of any kind should not be administered. Unconsciousness should, under such circumstances, never be induced and narcotics should not be administered. After tracheotomy, general anesthesia, including ether or chloroform, may be administered through the tube if there is no involvement of the lungs. If dyspnea is well established and severe, there may not be time even to infiltrate a local anesthetic, in which case an emergency tracheotomy must be done without anesthesia.

It is obvious that when there is trismus of even moderate degree it is not possible to introduce a laryngoscope or bronchoscope, and free breathing may be established with certainty only by tracheotomy. Dypsnea is a very positive danger signal in all such cases. My custom has been to establish free breathing by tracheotomy under local anesthesia, after which general anesthesia may be safely employed, given through the tracheotomy tube. Intravenous anesthesia with pentothal sodium is now being extensively used for many conditions formerly limited to local anesthesia. With a free airway established by tracheotomy this intravenous anesthetic offers the solution of the problem. It may be used in cases in which there may be some deep respiratory infection and in elderly people. I have not used it in children. In moribund cases no anesthesia of any kind should be used.

In cases with marked submaxillary and sublingual swelling and with swelling and crowding of the tongue, we have successfully and safely administered general anesthesia, but only after providing a good airway with a pharyngeal or nasal breathing tube. One must be certain that there is no edema of the larynx. A speaking voice of good quality is fairly good evidence that there is no laryngeal

edema. Anesthetics which are not usually followed by vomiting should be chosen, such as cyclopropane. The nasal and pharyngeal tubes should remain in place until after recovery from the anesthesia.

When there is dyspnea, sedatives and narcotics should not be administered for any reason until the dyspnea has been completely relieved. After the operation and with a good airway demerol may be administered as a sedative. It does not depress the respiratory center.

The use of local anesthesia in deep neck infections has many advocates. Ideal anesthesia may be obtained by injection of the cervical nerves at their points of exit from the cervical spine by the posterior approach. By this route the needle does not traverse any inflamed tissues. A good regional anesthesia is obtained. This presupposes the presence of one skilled in the technique of paravertebral injection.

If there is no inflammatory involvement of the parts good anesthesia of superficial areas may be obtained by blocking the branches of the cervical plexus at the posterior border of the sternomastoid muscle. If, however, there is inflammatory involvement of the part to be injected, it is difficult to obtain anesthesia of anything but the skin. Injection into inflamed tissues should be avoided, for it traumatizes already inflamed tissues, thereby intensifying and spreading the inflammatory process.

To the patient, incomplete and inadequate anesthesia is a torturous and unforgettable experience.

421 HUGUENOT STREET.

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The Scientific Papers of the American Broncho-Esophagological Association

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LXI

ADENOMA OF THE BRONCHUS

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Benign adenomas of the bronchus present an interesting group of tumors about which there is still some disagreement concerning the exact histologic classification. No attempt will be made here to enter into this question; we wish to report what seems to us an unusual and interesting case of the occurrence of one of these tumors.

The patient was a white female, eight years of age, who was first seen by one of us on February 26, 1946. At that time the following history was obtained from the mother. She stated that the girl had been subject to frequent respiratory infections since the age of three years. Because of these repeated episodes, roentgen-ray examinations of her chest had been made at intervals of approximately six months since that time. Each examination had been reported as normal. In addition to the repeated episodes of respiratory infection, the child had had audible wheezing almost every night for the past five years. This wheezing was not noticed during the daytime. It was considered to be asthmatic in origin, since the mother was known to have severe asthma. On December 24, 1945, the child was admitted to the hospital with a temperature of 104° F., cough, and dyspnea. A diagnosis of lobar pneumonia was made and she was treated with penicillin and sulfadiazine. This was continued until January 15, 1946, when she was permitted to go home. The

fever had subsided, but the cough and wheezing continued. However, she was readmitted on January 22, 1946, with a recurrence of the fever, the elevation being recorded at 105° F. Her illness was again diagnosed as lobar pneumonia and chemotherapy was instituted. This was continued until February 26, during which time the elevation of temperature continued. During this interval, roentgen-ray examinations showed an atelectasis and consolidation of the entire left lung.

On February 26, 1946, one of us was called to see the patient and advised a bronchoscopic examination. This was done and a tumor, originating from the left upper lobe bronchus, was visualized and removed. Following this, the fever subsided with only occasional elevations until March 15, 1946, at which time she was allowed to go home. There has been no wheezing since the day of the bronchoscopy.

The child was admitted to another hospital under the care of the other one of us on May 1, 1946. During the first week of this hospitalization, she had a daily elevation of temperature of 100° to 101° F. and a leukocytosis. She was given penicillin parenterally, and after the first week the temperature was essentially normal.

Roentgen-ray examination of her chest at this time showed a persistent hazy density extending out from the hilum into the left upper field.

Another bronchoscopy was done. There was a slight roughening of the mucosa just inside the left upper lobe bronchial orifice, but otherwise no evidence of the tumor was seen. Subsequently, iodized oil was instilled into the left lung and this demonstrated a rather extensive bronchiectasis of the left upper lobe, the lower lobe being essentially normal.

The child was discharged from the hospital on May 25, 1946, apparently symptomatically relieved. A communication from the mother in January 1947 stated that the child had been well since discharged and had gained 15 pounds in weight. A comparative chest examination showed essentially no change in the appearance of the left lung field.

The question of the advisability of surgical removal of the left upper lobe for the bronchiectasis was discussed with the parents, but they were not agreeable to the idea.

A case of benign adenoma occurring in an eight-year-old female is reported. From the history it seems reasonable to suppose that this tumor had been present since the age of three, causing symptoms of partial bronchial obstruction which were erroneously diagnosed as allergic because complete diagnostic studies were not done.

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707 MEDICAL ARTS BUILDING.

LXII

SCLEROMA: A CLINICO-PATHOLOGICAL STUDY OF SEVEN CASES IN ONE FAMILY

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and

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Levine, Hoyt and Peterson[1] have reported a series of cases of scleroma in an etiological study. Levine and Hoyt[2, 3] have also published comprehensive reports on the serology and the diagnosis of the disease. The present paper is presented to amplify the clinical description, to describe the therapy attempted, and to supplement the series with one possible additional case in the same family.

At the 1942 meeting of the American Medical Association, before the Section on Laryngology, Otology and Rhinology, Cunning and Guerry[4] reported a series of six cases of scleroma observed at the Manhattan Eye and Ear Infirmary. In a comprehensive study, they have collected from the literature since 1893, 102 cases occurring in the United States and Canada. Sixteen of these persons were born in the United States. During the past five years Dixon,[5, 6] Dill,[7] and Kellert,[8] each added a case, but these persons were born in Europe. Our patients have never been outside the continental United States.

A review of cases that have been reported shows that in a number of instances the writers give descriptions of the late stage of the disease in which either the external nose and the upper lip had undergone a grotesque monstrous deformity or there was a stenosis of the larynx resulting in progressive asphyxia. As might be expected, the histologic study is usually limited to a description of the tissue changes that were observed in late lesions.

From the Departments of Otolaryngology, and Pathology and the Institute of Experimental Medicine, College of Medical Evangelists.

In the present series each case shows a different period of development of scleroma—from the incipient, subclinical, asymptomatic state in a 12-year-old girl, to the final deforming scleroma of the typical textbook description in the father and the son. Each patient showed a strong positive reaction to complement fixation, and there was abundant growth of Frisch's bacilli on culture of secretions from the respiratory tracts.

REPORT OF CASES

CASE 1.—L. M., farm laborer, aged 38, born in Colorado, was admitted to the White Memorial Hospital on March 12, 1947. His grandfather was a native of France and his grandmother was a Spaniard. They emigrated to the United States early in the nineteenth century and raised a family in Colorado. His father lived until 1939 when he died at the age of 87. His mother was a full-blooded Sioux Indian from North Dakota. The patient had lived continuously in Colorado until he was 33 and then moved to California in 1942. He has two brothers and two sisters, all living and well. He married a woman of Mexican ancestry from New Mexico at the age of 17, and separated from her seven years ago. There are two sons and four daughters in his family. The oldest son (Case 2), aged 21, is married and has a child. He maintains his own home. Until March 1947, the remainder of the family including four girls, J. M., aged 19 (Case 3); C. M., aged 18 (Case 4); M. M., aged 15 (Case 5); R. M., aged 12 (Case 6); and the youngest son, R. M., aged 9, lived in a moderate sized trailer under extremely congested conditions.

The patient has spent most of his life on a farm. At the age of 27 he broke his nose, being kicked by a horse. No treatment was given. He dates the beginning of his nasal disorder to this accident. In the beginning there were symptoms of nasal catarrh with a varying amount of nasal discharge which at times was foul. Gradually, he lost the sense of smell. Nasal breathing became increasingly difficult. For the past eight years he has not been able to breathe through the nose.

In 1936 he was admitted to the Colorado General Hospital on account of nasal obstruction. The record indicated[8] that the nostrils were occluded by a white tumor and a small amount of white discharge on each side. The tumor invaded also the anterior pillars of the tonsils. A diagnosis of scleroma was made after a period of investigation. From February 24, 1936, to May 29, 1937, he had

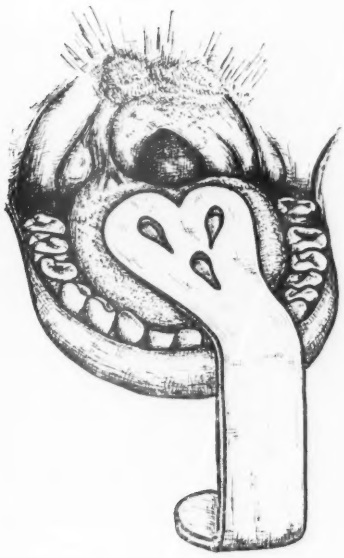

Fig. 1, Case 1.—The appearance of the father's throat. The soft palate has undergone scleromatous deformity, the uvula being buried in the mass of scarred tissue. The pharynx is reduced to about one-third of the normal size. Note the fibrous band stretched between the base of the tongue and the right tonsillar fossa.

received several courses of x-ray therapy. On August 7, 1937, a right Caldwell-Luc operation was performed. Histological study indicated the involvement of the antrum by scleroma. The left antrum was not entered surgically. He was discharged from the hospital on August 16, 1937. About a year and a half after the last x-ray treatment, he developed bilateral cataract. For the past five years he has worn glasses, but his vision is greatly impaired. He eats and sleeps well. There is no pain. His normal weight is about 160 pounds. At the present he weighs only 138 pounds.

Examination: The patient was 5 feet, 6 inches tall, dark-skinned, with a sharp high nose. He was coöperative and intelligent. He was well developed but poorly nourished. His voice was clear but he spoke with tones characteristic of one with nasal obstruction. The nasal pyramid was grossly displaced to the right. The nasal septum was also deflected to the right. The nasal mucous membrane was uniformly thickened, but smooth and salmon pink in color. No

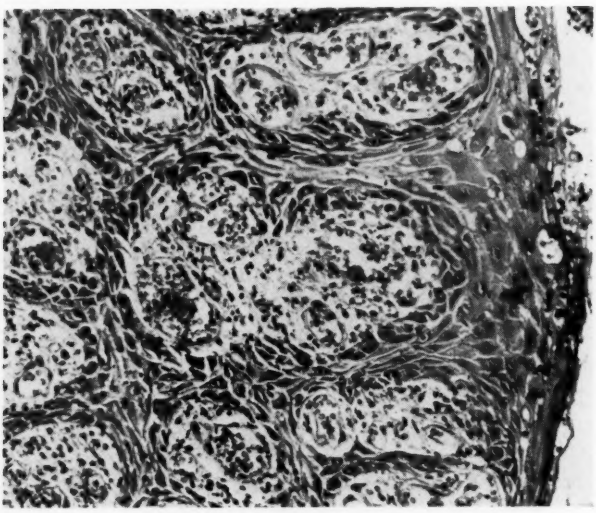

Fig. 2, Case 1.—The section removed from the pharynx, showing an irregular pattern of stratified squamous epithelium with slender anastomosing rete pegs, projecting into the granulation tissue in the stroma. These papilli contain numerous Mikulicz cells, a few neutrophils, lymphocytes, plasma cells, and occasional hyaline bodies. (X 117)

crustation was present. The posterior choanae were completely occluded. The granulomatous infiltration transformed the soft palate into a mass of hard cartilage-like scar tissue, and the uvula was pulled upward (Fig. 1). The lateral pharyngeal space was completely obliterated by a fibrous mass. The posterior pharyngeal wall was greatly thickened and fibrosed. The nasopharynx was reduced in size to about one-third of the usual capacity, barely permitting insertion of an index finger. The patient wore a complete upper denture. The lower teeth were free from caries. The tonsils were atrophied. The pillars of the tonsils were widely separated because of contraction of the lateral pharyngeal muscles. A fibrous band stretched from the base of the tongue to the right tonsillar fossa. The entire larynx appeared to be pulled upward. The laryngeal mucosa was moderately congested. The motility of the cords was fair. The granulomatous infiltration extended well into the posterior wall of the trachea. Both ear drums were thickened. The cervical glands were not enlarged.

On March 12, 1947, under pentothal sodium anesthesia, a plastic operation on the posterior choanae was performed, a curtain-like fibrous wall on each side being removed. Following the operation the patient received daily 2 grams of streptomycin intramuscularly and ½ gram by aerosol inhalation in 4 divided doses for 10 days. The Frisch bacillus could not be recovered from his lesions after the sixth day of treatment.

Figure 2 shows a biopsy specimen removed from the warty growth on the posterior pharyngeal wall. The surface epithelium varies from a thick eight- or ten-cell covering to a thin one- or two-cell layer. The squamous epithelial cells are quite large with small dark-staining nuclei. Intercellular bridges are prominent. A rete-like structure of epithelial network encloses cross sections of papilli. The papilli show a delicate structure containing thin-walled capillaries surrounded by characteristic Mikulicz cells, a few lymphocytes, plasma cells, and occasional Russell bodies. This is a typical picture of scleroma.

Most characteristic of this lesion were the cells first described by Mikulicz[10] which bear his name. They probably originate from mononuclear cells. The Mikulicz cell appears as a large clear vacuole surrounded by a narrow zone of cytoplasm. If the plane of the section happens to pass through the nucleus, a thickening in the zone of the cytoplasm will be seen. This will contain a small pyknotic nucleus. In any given section nuclei will not be shown in the majority of Mikulicz cells, as a thin section passes through only a few of the nuclei. When placed side by side the vacuolated cells resemble lace. Hence they have been named foam cells or lace cells. Within these cells are bacilli of Von Frisch, and sometimes the organisms are seen extracellularly at the rupture of these cells. These bacilli are encapsulated, gram-negative, short, plump rods with rounded ends. Russell bodies are collections of homogenous eosinophilic hyaline substances. They are usually round in shape and uniform in size. Dark-staining, roundish to oval nuclei are found either in the center of these cells or in the periphery. Sometimes the nucleus is absent, depending on how the cells are cut in the preparation. Russell bodies are supposed to have originated from mononuclear cells,[11] but others consider that they originate from plasma cells.[12]

Another specimen was taken from the scarred area of the choanae. It showed heavy eosinophilic collagenous fibers occurring in irregular arrangement, most of the secretion being relatively acellular. In some areas there was a dense collection of pyknotic nuclei. These nuclei were closely packed and distorted. These cells were

hard to identify, but probably they originated from connective tissue. The epithelium occurred in scattered islands on the surface and the lining of depressions. The epithelium was ciliated on the surface and had multiple layers. This section is a good example of scarring in scleroma.

CASE 2.—L. M., aged 21, married, a truck driver, the oldest of the six children, was the first in this family that came to our observation. He had lived in Colorado until 1942 when he moved to California at the age of 16. The patient was first admitted to the service of Dr. John E. Peterson in the Medicine Clinic of the Out-Patient Department of the White Memorial Hospital. Later he was referred to the Otolaryngological Department in January 1946. The diagnosis of scleroma was confirmed by bacteriological studies and complement fixation test made by Levine and Hoyt. The patient has had nasal obstruction of about four years' duration. He complained of hoarseness, postnasal discharge, and a cough with expectoration for about a year. In 1940 he broke his nose while playing football, for which he received no medical treatment. About a year after the accident he began to have difficulty in breathing, which has grown progressively worse. Since coming to California he was seen in two other hospitals where he received antibiotics in one and a series of six x-ray treatments in the other. Some relief in nasal obstruction was obtained.

On the initial examination the nasal passages were obstructed by granulation tissue 2 cm. within the nasal vestibule. Externally, the nose was slightly deflected to the right on account of the unreduced fracture. The base was widened and looked like a nose that had suffered from a chronic bilateral ethmoiditis. The nasal septum was deviated to the right. The turbinates were atrophic. The entire nasal mucous membrane was coated with thin yellowish gray-green crusts which emitted a distinct odor not unlike that of ozena. The crust came off with some difficulty causing a fine capillary oozing from beneath. His tonsils were small and coated with whitish flecks. The teeth and gums were healthy but his breath was foul, sour and stale. The tongue moved freely. The tip of the uvula was drawn upward and curled into a mass of scar tissue and was only visible on posterior rhinoscopy. The lateral pharyngeal spaces were obliterated by thickened fibrous tissue. The hard whitish cartilage-like scar tissue stretched across the thickened posterior pharyngeal wall as the result of cicatrization This portion was covered with a dry glue-like discharge. The choanae were greatly reduced in size, the left one barely permitting a small metal probe to pass. The laryngeal

Fig. 3, Case 2.—The appearance of the pharynx. Note the granulomatous mass filling both tonsillar fossae. The uvula is drawn upward and hidden behind the scarred mass.

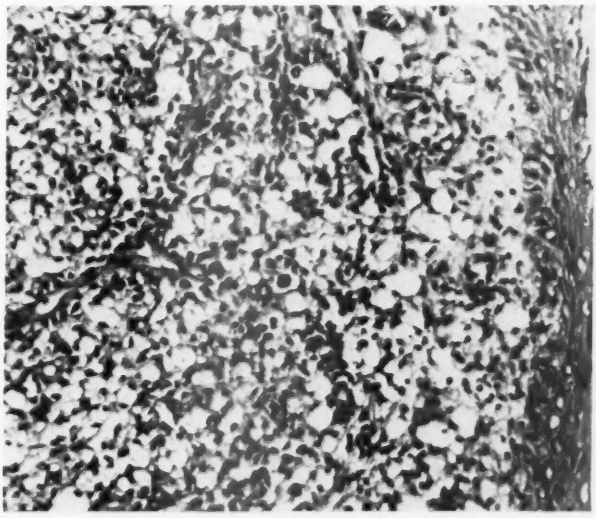

Fig. 4, Case 2.—The section removed from the nasal floor showing metaplastic squamous epithelium and numerous Mikulicz cells surrounding the network of capillaries. A hyaline body (Russell's) is well shown in the center. There are numerous round cells, plasma cells, and polymorphonuclear leucocytes throughout the stroma. (X 117)

mucous membrane was diffusely thickened. The voice was husky and had a distinct nasal twang. The motility of the vocal cords was not impaired. His hearing acuity was not reduced. There was no cervical adenopathy. He received 10 grams of streptomycin in divided doses. Following this the organisms were greatly reduced in number but were not entirely irradicated.

On November 12, 1946, a plastic operation was performed to reëstablish a more normal choanal passage, using the technique advocated by Kazanjian.[13] He was seen on February 24, 1947, and had gained 22 pounds in weight since the plastic operation. On May 5, 1947, a diagnostic bronchoscopy was performed. The mucous membrane of the trachea and bronchi was uniformly thickened and salmon-pink in color. Arrangement is being made to continue treatment.

Figure 4 shows the section removed from the nasal floor. The stratified squamous epithelium stretches over the granulation tissue which appears vacuolated in some areas, but the bulk of it is composed of numerous Mikulicz cells, and a network of small capillaries around which are cords of wandering cells. There are a few neutrophils, lymphocytes and plasma cells. Russell bodies, some with eccentric small nuclei, are relatively numerous.

A section of the specimen removed from the carina shows that the ciliated epithelium has undergone squamous metaplasia and is enormously thickened. Rete-like prolongations project into the submucosa which is composed of granulation tissue. There are numerous capillaries. The cellular elements consist of neutrophils, lymphocytes and plasma cells. In some areas the epithelial layers are infiltrated with leucocytes. Numerous Mikulicz cells with eccentric nuclei are surrounded with plasma cells, lymphocytes and degenerated polymorphonuclear leucocytes scattered throughout the granulation tissue. Characteristic Russell bodies are not present.

Case 3.—J. M., aged 19, single, the oldest of the four daughters in this family, is a native of Colorado, where she lived until 1942 when she moved to California. She owns and manages a restaurant with her sister (Case 4). Since coming to California she has had almost continuous head colds with purulent nasal discharge. This she attributed to the change of climate. She complained of a stuffy sensation in the nose as though not being able to get enough air; also that expectorated sputum was purulent. Her throat has been intermittently sore. She has been conscious of a foul odor from her nose and throat since the beginning of the present trouble. At the age

of five she had measles; tonsillectomy at 11; diphtheria at 13; and suppurative otitis media at 15. She has worn glasses since she was nine and had recurrent attacks of blepharitis. The sense of olfaction was slightly impaired. Otherwise her general health was excellent.

Examination: The patient was visited on February 24, 1947. There was no external deformity of the nose. The nasal mucosa was dry. The turbinates were shrunken and hidden beneath thick crusts of grayish-green exudate. Much similar discharge was found in the nasopharynx. After the crusts were removed the posterior pharyngeal wall presented a beefy appearance. Her teeth were well cared for and the gums appeared healthy. The right tonsillar fossa was filled with recurrent growth. The left fossa was clean. The larynx was not congested and there was no impairment of phonation.

CASE 4.—C. M., aged 18, single, a native of Colorado, a part owner of the restaurant, came to California with her family in 1942.

At the age of seven she developed pneumonia complicated with empyema which required incision and drainage. As a child she had had repeatd sore throats and was relieved after a tonsillectomy at the age of 11. She did not remember the exact mode of onset of her present trouble. It began as a nasal catarrh associated at first with watery nasal secretion which became later more tenacious and malodorous. She has blown her nose a great deal and feels as though she has had a continuous cold. She was anemic. Her voice was distinctly husky with a nasal twang.

Examination: This patient was seen at the same occasion as Case 3 on February 24, 1947. Her nasal cavities were wide open. The posterior pharyngeal wall was readily visualized as the turbinates were atrophied. A thin odorous crust of yellow-gray color was observed over these turbinates. A similar crustation was noted on the pharynx. The laryngeal mucosa was moderately injected but the vocal cords moved freely and its mucosa was faintly colored. There was no cervical adenopathy.

Figure 5 shows the biopsy specimen removed from the anterior tip of the left middle turbinate from Case 3. The histologic section from the same area in Case 4 indicated almost identical tissue changes.

The epithelium is partly stratified squamous and partly stratified columnar. Within the stroma there are a number of nasal glands which are inactive and whose lumina are wide open without containing mucus, giving one an impression of an atrophic, shrunken structure. The stroma is slightly edematous, but it does not present

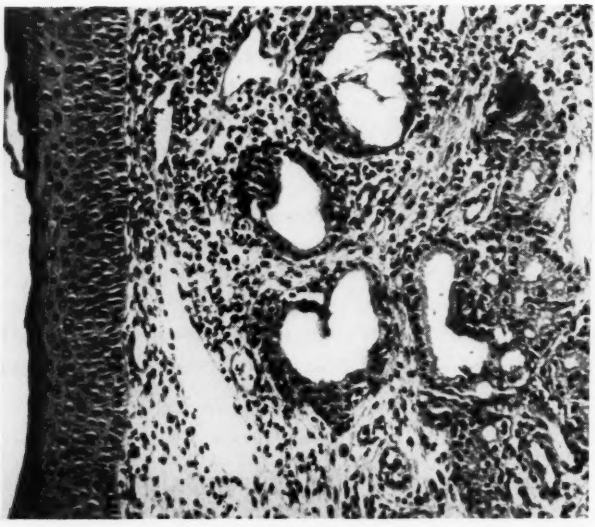

Fig. 5, Case 3.—Section removed from the anterior tip of the left middle turbinate. The epithelium is stratified squamous. Within the stroma are numerous nasal glands which are inactive and whose lumina are wide open without containing mucus, giving the appearance of an atrophic shrunken structure. (X 117)

a typical picture of granulation tissue. The fibroblasts are inactive and their nuclei are shrunken. The chief cellular elements are small round cells, a few plasma cells and scattered neutrophils. The characteristic histocytes are present but they are not numerous. The picture is essentially that of chronic inflammation.

Clinically, Cases 3 and 4 are in the dormant or inactive state.

Case 5.—Mary M., aged 15, a high school student, was born in Colorado. She moved with her family to California in the summer of 1942. She also was seen on February 24, 1947. Ever since she came to California she has had a continuous nasal obstruction with much purulent malodorous discharge. Whether her nasal catarrh began in cold weather or after her residence in California, she was not quite certain. For several months her sisters have noted that her pillow was being increasingly stained yellow on account of the overflow from her nose. Her sense of smell was poor although she could taste most foods.

Examination: The patient was well developed and nourished. There was no external evidence of any disease. Her breath had a distinct odor. A creamy discharge flooded the nasal floor on each side. Both nasal passages were obstructed. This overflow left a reddened streak on the nasolabial fold and the mucosa was pink and bled easily on manipulation. The septum was thickened and was slightly deviated to the right. The turbinates have undergone granular infiltration and were thickly coated with greenish-yellow exudate. The posterior pharyngeal wall was coated with exudate originating from the nose. No scleromatous changes were shown on the palate and nasopharynx. The tonsils were small and submerged. The teeth and gums were in a healthy state. The laryngeal mucosa was moderately congested. The vocal cords moved freely. The voice was clear. No palpable cervical glands were present. Of the three girls this patient showed the most active suppurative infection. Specimens were taken from the right nasal vestibule, the anterior tip of the left lower and of the middle turbinates. On May 22, 1947, a diagnostic bronchoscopy was performed by Dr. John Cremer, assisted by Dr. M. O. Dart of Denver. The patient reacted well to the procedure. The tracheobronchial mucous membrane was moderately inflamed, the right bronchus much more than the left.

A section of the specimen from the floor of the nose shows the surface epithelium ulcerated in some areas. Upon it lies fibrinopurulent exudate. The crust has largely broken off from the underlying tissue. The epithelium consists of four or five layers of cells which have transformed into the squamous type. This epithelium overlies the granulation tissue. Their nuclei are large, oval or rounded in shape, many of them containing nucleoli one-fourth to one-fifth the diameter of the nucleus. Mitosis is frequently observed. The granulation tissue is more or less edematous. There are numerous blood vessels and capillaries presenting hyperplasia of endothelial cells. The stroma is densely infiltrated with wandering cells which is composed of numerous plasma cells, some lymphocytes and neutrophils. There are a large number of mononuclear cells which are most numerous about the blood vessels. Some of the mononuclear cells are vacuolated and foamy in appearance, but these are not entirely characteristic of Mikulicz cells. The section is indicative of an earlier lesion of scleroma—appears more like a nonspecific granulation tissue.

CASE 6.—Rita M., aged 12, a ninth grade student, was born in Colorado and is the youngest daughter in the family. She came

to California with her family in 1942. The patient was seen on February 24, 1947.

Examination: The patient was well developed and nourished. The tonsils were moderately enlarged. The teeth and gums were in good condition. Her nasal mucosa was not congested but appeared dry, as though it was in an incipient state of atrophic rhinitis. There was no granulation or crusting. Her breath was not foul. The swab specimen taken from her nose and nasopharynx yielded an abundant growth of Frisch's bacillus on culture. A biopsy specimen removed from the middle turbinate on May 20, 1947, indicated a possible early metaplasia of the surface epithelium. The stroma appeared essentially normal.

These patients were instructed to return to the clinic in April 1947. In this they failed. Later we learned that the girls had left California early in March to visit their mother in Denver. On May 9, 1947, the three older girls were rechecked. On May 20, 1947, the youngest daughter and the mother were examined. Dr. Dart's report of the mother's examination is as follows:

CASE 7.—"A. M., 37-year-old Mexican, was born in Taos, New Mexico, and moved to Colorado early in life where she has continued to live until the present. Her mother died at the age of 36 from an unknown cause. Her father died in 1931, his age and the cause unknown. She was married at the age of 16, and separated from her husband (Case 1) in 1940. She has had an impairment of the sense of smell for about two years. She has never had any nasal obstruction, nasal discharge, postnasal discharge or cough. She does not blow her nose. She has never received any treatment for any nasal trouble.

Examination: "The nose has all the characteristics of an atrophic rhinitis. All turbinates are markedly atrophied, especially the middle turbinates. There is a lot of moist yellow crustation and purulent discharge, especially on the turbinates. There is no odor to the discharge. It looks just like Jennie's and Cecelia's noses (Cases 3 and 4). The tonsils are small. The pharynx is negative. There is no evidence of postnasal discharge. The ears are normal. There are no cervical glands palpable. The skin of the face and head is normal. The remainder of the examination was normal."

From her pharyngeal smears there was recovered in culture an abundant growth of Frisch's bacilli. The complement fixation test was positive for scleroma. The biopsy specimen removed from the

middle turbinate indicated granulomatous changes very similar to those observed in Cases 3 and 4.

DISCUSSION

This series suggests that scleroma is contagious and transmissible by long contact under poor hygienic environment. However, Levine and Hoyt prefer to be noncommital as to its communicability. No facts are available regarding the mode of transmission. Most likely the father is the original source of infection. Koch's postulate, as in some other granulomata, has never been fulfilled. The only member of the family whose nasal culture and complement fixation test have been consistently negative is a nine-year-old son.

The laboratory diagnosis of scleroma, including bacteriological studies of Klebsiella rhinoscleromatis and complement fixation tests using preparations of this organism as a test antigen, is being reported by Levine and Hoyt.

Chamberlain[14] has attempted to classify the changes in the mucous membrane into three different stages. These are: (1) the primary nasal catarrh with increased secretion and subsequent crustation; (2) proliferation and tumefaction; and (3) the final sclerosis and the deforming scar formation. Morrison[15] states that the overlapping of these three stages is clinically observed and histologically noted in specimens removed from various parts of the respiratory tract of the same individual, especially in the second and third stages. This is well illustrated in our own cases.

Histologic diagnosis of scleroma in the early stage is difficult (Case 6). The inflammatory exudate produces an interstitial edema and a varying degree of infiltration of leucocytes and plasma cells. Macrophages are numerous and diffusely distributed. Suppurative rhinitis (Case 5) is later followed by atrophic changes (Cases 3, 4 and 7). The picture is that of nonspecific chronic granuloma. The distinguishing feature of scleroma is best observed at the site of proliferation but not in scarred areas (Cases 1 and 2). The ciliated columnar epithelium is transformed into a thick squamous type. Rete-like prolongations, some thick and others thin, penetrate deeply into the submucosa, often surrounding the underlying granulomatous tissue. Later the acute inflammatory reaction subsides. Only macrophages and degenerated plasma cells are observed. This is most conspicuous around the area of the epithelial prolongations and anastomosis. In a more advanced stage the macrophages suffer chemical changes. The cytoplasm appears vacuolated and reticulated.

The nucleus becomes pyknotic. These are Mikulicz cells. Mikulicz cells are also seen in glanders, bubonic plague and leprosy. Within them are bacilli of Von Frisch, and the organisms are seen extracellularly at the rupture of these cells. There is also seen so-called Russell bodies. These are spherical, eosinophilic bodies which originate from the degenerated mononuclear cells. Neither bone nor cartilage is involved.

Both father and son believe the onset of their nasal disorder immediately followed trauma with fracture. Each had atresia of the choanae for which a plastic operation was performed. Both showed markedly similar lesions and destructive deformity of the soft palate and of the pharynx. This might show that each strain of Frisch's bacilli (and there are nine) shows a particular predilection for a certain area in the respiratory tract. None of these seven showed a skin lesion.

Roentgen therapy, which has been reported to be beneficial by some writers, was ineffective in both father and son. Sulfanilamide and sulfadiazine also failed. Streptomycin in adequate doses appears to be the most potent drug at our command. We used daily .5 gram of aerosol inhalations and 2 grams intramuscularly in six divided doses. Organisms could not be discovered from the throat after the sixth day of treatment in Case 1. Case 2 received 10 grams of the drug. At the last visit on May 5 he still harbored some organisms.

We agree with Dixon that the time element is so important in the permanent cure of such disorders that it is much too soon to state the final outcome under the present regimen. But at present, both father and son have never felt better. Plans are being made to treat the other members of the family.

SUMMARY

1. A detailed study on seven cases of scleroma from one family is presented.

2. Histologic changes in the mucous membrane of the respiratory tract—from the incipient nasal catarrh, followed successively by suppurative rhinitis, atrophic rhinitis, chronic granuloma to the final deforming scleromatous scar—are shown.

3. Of the various therapeutic measures tried streptomycin in an adequate dose is the most promising.

4. A diagnosis of scleroma can be made by trained bacteriologists and serologists long before histologic changes are recognized.

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LXIII

FORTY-FIVE CASES OF PEANUTS REMOVED FROM THE LOWER RESPIRATORY PASSAGES

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Since 1919, much has been said and written about the aspiration of peanut kernels into the lower respiratory passages, the pathologic changes observed in those cases and the many happy and unhappy end results obtained in dealing with that category of foreign body.

Paterson, Jackson and Spencer introduced into bronchology the term "arachidic bronchitis" to describe the specific type of bronchitis observed when a peanut is inhaled. In 1923, Chevalier Jackson, Tucker and Clerf reported on "Arachidic and Other Forms of Vegetal Bronchitis." In 1930, Heatly and Clauson brought to the attention of the members of this Society some experimental studies in the rabbit with pathological examinations and chemical analyses of different types of peanuts, drawing interesting conclusions concerning peanuts inhaled by children. The following year Stiles presented some observations on a series of 50 cases of vegetable foreign bodies in the trachea and bronchi. To my knowledge the last paper on that subject read before the American Broncho-Esophagological Association was by Hill and was entitled "A Consideration of Some of the Causative Factors of Vegetal Bronchitis."

The purpose of my presentation is not to repeat what has been said before but merely to submit a series of 45 cases of peanuts in the lower air passages which I had the opportunity to treat, up to May 1947. Since then some 15 additional cases were treated in the Department of Broncho-Esophagology of Ste.-Justine Hospital, Montreal, Canada. I thought that to present a brief synopsis of the whole group would be more conclusive on points of interest about the anamnesis, frequency, age, symptoms, location, x-ray findings, clinical course and treatment of these vegetal foreign bodies as seen in the Canadian provinces of Quebec and Ontario.

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This group of foreign bodies represents 60 per cent of a total of 75 foreign bodies of all sorts removed from the larynx, trachea or bronchi during the same period of time, and approximately 16 per cent of the total of 270 foreign bodies extracted from the upper food passages and from the lower air passages by one of the direct endoscopic methods. Moreover, in the month of March 1947, we removed one or more fragments of peanut kernels from seven children. This means that there occurred during this month alone more than 16 per cent of all our cases of peanuts and 10 per cent of all our cases of foreign bodies removed from the lower air passages for a period of nine years. I feel that this occurrence can be attributed to two factors: namely, a greater abundance of peanuts on a postwar market, and a much greater knowledge on the part of general practitioners and pediatricians who are now more aware of the possibility that a foreign body may be the cause of distress in breathing and explain the onset of a rapidly developing pathological condition of the bronchi or of the lungs.

In our group, two adults aspirated pieces of peanut kernel: a married woman 46 years of age and a single woman 33. If we exclude these two persons the age scale is from 8 months to 10 years; 37 children (slightly more than 80 per cent) being from one to three years of age. There seems to be no doubt that parents, relatives or friends are not inclined to give peanuts to a child under 12 months. Children between one and three years of age cannot or can only with difficulty chew up these vegetal kernals, and there are many other factors met with frequently in children of that age, such as crying or falling down with food, candies, toys or peanuts in their mouths. Ninety-two per cent of the peanuts have been removed in children. This is close to the 99 per cent of C. Jackson and C. L. Jackson and their co-workers.

Thirty patients, or sixty-six per cent, had a definite history of having choked on a peanut. Five had no previous history of a foreign body and such was not even suspected at time of bronchoscopy. Of these five, one had negative x-ray findings, two had atelectasis of the base of the lung, one had only a peribronchial thickening and the fifth one had a generalized bronchopneumonia with atelectasis and consolidation at the right base. One with atelectasis at the left base was 16 months old and had been in our hospital for 45 days before a bronchoscopy was thought of; he had been ill for the previous two months. Recovery was complete 12 days after removal of the peanut kernel and repeated bronchoscopic aspirations. At no time did he show signs of laryngeal distress, but his general condition was

poor; he had a toxic appearance and a high spiking fever. The 16 other patients had no definite history of a peanut but a history of a sudden onset of the disease, in most cases with choking, suspicious of the inhalation of some foreign body. Of course, I mean it was suspected, not by the family members or relatives who were totally unaware of the accident but by the family physician, the radiologist or the bronchoscopist. By correlating the details of the history, the clinical signs, and the x-ray findings, the inferential diagnosis of a vegetal foreign body could be made. However, in the four bronchoscopically discovered peanuts, the history, symptoms and x-ray findings did not permit a positive differential diagnosis between infectious bronchitis, asthmatic bronchitis, bronchopneumonia, emphysema or unresolved pneumonia. Bronchoscopy alone solved the problem.

Thirty pieces of peanut kernels were inhaled into a right bronchus and 11 into a left bronchus; only 3 anchored in the trachea above the carina. This ratio of nearly 3 to 1 in favor of the right side for the location of peanuts is nearly the same for all bronchial foreign bodies and finds plausible explanations in the anatomy and size of the main bronchi. The angulation and the much smaller caliber of the left bronchus prevent a foreign body of a certain size from taking that route. Moreover, the negative pressure in the right lung and the flow of air through the broader and more direct stem bronchus is certainly stronger on the right side than on the left. In one case only, did we find a piece on both sides; one in the left main bronchus and one in the right lower lobe bronchus. The 18-month-old male arrived in a critical condition with marked indrawing, cyanosis and exhaustion. The temperature ran from 102° to 105°; there was atelectasis and consolidation at the right base plus obstructive emphysema of the left lung. The foreign bodies had been inhaled 17 days previously. This is one of the few cases in which we noticed an hyperleucocytosis: 28,000 and 38,000 white blood cells were reported. A tracheotomy had to be done on the fourth day. Despite intensive penicillin therapy and repeated bronchoscopic aspirations, the child died on the thirtieth day after admission. At autopsy there was atelectasis and consolidation of the right middle and lower lobes plus emphysema and bronchial obstruction of the left lung by crusts and membranes.

In our patients the pieces of peanut kernel were usually big enough to stop and anchor in a main bronchus; most of them anchored in the proximal portion of the right bronchus and close to the upper lobe bronchus. They moved slightly to and fro with the air current except when they were inbedded in considerable gran-

ulation tissue. We observed this in nearly all of the 13 cases of atelectasis of one or more lobes. When the foreign body moved with breathing, we could easily correlate the findings with previous signs of clinical and radiological obstructive emphysema. Only in a few cases of peanut kernal obstruction, did we witness a substantial shifting of the heart and mediastinum towards the unobstructed side with expiration, known to all in marked obstructive emphysema of a whole lung. I feel that as long as the air flows to a certain extent into the upper lobe, even thinly, there is enough inflation to balance the pressure of the nonobstructed lung when it is fully distended at the end of inspiration. This is especially true of the right upper lobe; its lobar bronchus is at the level of the carina or slightly lower, or even above that plane in certain cases.

Only in 7 cases were the foreign bodies in a lower lobe bronchus: 4 in the right lower, 2 in the left lower, 1 in the right lateral basal bronchus. This is easily understandable when one knows how tiny those bronchi are in children.

Our oldest patient, a married woman of 48 years, had partial obstruction of the right lower lobe bronchus for ten months. In the other adult, a female of 33 years of age, the peanut kernel had obstructed the lateral basal bronchus of the right lower lobe for seven days. The corresponding pulmonary segment was atelectatic. That is the only case in which I have clearly visualized the peanut kernel in a segmental bronchus; the segmental atelectasis also was clearly mapped on the roentgenograms.

The peanut kernel was located low in the trachea in 3 cases, one for as long as four to six weeks; only one required immediate removal by emergency bronchoscopy. This was in the oldest of the three (six years of age) who had inhaled the foreign body less than 24 hours before; the peanut kernel was very large. The age of the two others was two years and three and a half years. There was marked asthmatic wheezing, rattling and a juicy cough, many noisy respiratory rhonchi but nothing evident at the x-ray examination. All three recovered quickly.

The time of sojourn was from a few hours to ten months. Two months in a child of 16 months was the longest time observed for a piece of peanut to be lodged in a child's bronchus. There was marked consolidation and atelectasis of the left lower lobe. There were septic signs and symptoms rather than signs and symptoms of obstruction. The recovery was complete two weeks after removal. We have definite evidence that the time of sojourn has a bearing on

the patient's physical condition according to the size of the fragment of peanut and its location, and the age of the child. The child's life is more endangered when he is younger, when he has inhaled a bigger piece, and when the piece is more highly located. In other words, a big piece with a high location can kill in a short time, whereas a small piece in a low location can produce but few signs and symptoms and can show but progressive bronchopulmonary changes.

In many cases, the clinical symptoms were far from being classic; the patient sometimes has only slight cough or wheezing. Nevertheless, some asthmatic wheeze was present in almost all cases. On the average our children had but slight fever (between 99° and 100°) and their blood count was approximately normal, showing only a slight decrease in the blood corpuscles. We had evidence of hyperleucocytosis in only 8 cases: they were in the children with a toxic appearance who ran a temperature above 100° and had marked inflammatory edema on bronchoscopic examination. Surprisingly, children who had fever as high as 103° and 104° showed no pathological changes in their blood count. Here, I should like to emphasize that since the era of penicillin the temperature charts in these cases have changed considerably; the family physician often has used sulfonamides or penicillin for a few days before sending these patients to the hospital. That is why I feel that in our cases the average clinical picture was milder than the one described in the heroic days of bronchoscopy. It is also our impression that our cases could be controlled more easily by additional therapy than the cases reported previously. Penicillin injections in many of our patients had an immediate effect on their well-being before the foreign body was extracted; the temperature dropped suddenly even to normal; the breathing became easier and there was much less indrawing; however, that lasted only one or two days. We have noticed that it became easier to work because there was less inflammation and edema, and the secretions were more easily aspirated. It has become a routine at our clinic to inject 100,000 to 200,000 units of penicillin per day in fractional doses, starting immediately on the arrival of the patient and continuing after the extraction until recovery. If the arachidic bronchitis was due mainly to irritation by oleic and linolic acids of the peanuts as demonstrated by the experiments of Heatly, why is this type of bronchitis responding so quickly and so well to penicillin therapy? It might prove very interesting to repeat Heatly's experiments on rabbits adding injections of penicillin.

Up to now with the clinical observations of these 45 cases, I am inclined to conclude with Richards that the shape, bulk, location

and motion of the pieces of peanuts inhaled by my patients have played a definite role on the clinical radiological pictures according to the age of the patient and the type of obstructive valve encountered. The irritation seemed to be more of a mechanical than of a chemical nature; the infection, although being secondary, played with the obstruction one of the most important roles in the prognosis as shown by the rapidity of recovery or the findings at autopsies. The peanut was lodged in our adult patient for ten months and she had only a slight productive cough and peribronchial thickening of the right base. A child of 19 months had a piece of a peanut kernel in the left bronchus for three weeks; he presented only signs of obstructive emphysema and was discharged 36 hours after removal. A child of three and a half years who had a piece of peanut in the right bronchus for a month and another child of four years who had one in the right bronchus for two weeks also did not show any radiological sign or evidence of changes in the lungs. Many x-ray examinations have shown no definite sign of pathology even after the foreign body had been lodged for days and weeks. A negative fluoroscopic examination or a negative roentgenogram did not exclude the presence of a peanut in quite a few of our cases.

We had 5 deaths in this series, attributable to the age of the victim and to the severity of the obstruction, both irritative and infective. Fifteen patients were treated before penicillin had become available; of these, 3 died of lung complication. Since penicillin has been used, we have treated 30 patients with the death of only 2 and a complete recovery in 94 per cent of them. Should that be attributed to the type of peanuts inhaled or to the adjuvant action of our latest but wonderful drugs, or to both? That still remains to be definitely determined.

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LXIV

PRIMARY CARCINOMA OF THE TRACHEA REMOVED BY BRONCHOSCOPIC PROCEDURE

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Primary carcinoma of the trachea is rare. In a review of the literature up to 1938, Culp[1] reported 147 cases in which an accurate histologic diagnosis was obtained in only 87 cases. Because the diagnosis is generally established late in the course of the disease, successful treatment is usually impossible.

REPORT OF A CASE

The patient, aged 62, female, was first seen in June 1942. The chief complaint at that time was asthmatic wheezing with marked dyspnea. Her past history revealed that for the past three months she had undergone allergenic treatment for bronchial asthma. Not having responded to this form of therapy and her dyspneic condition having become progressively worse, she was referred for bronchoscopic study with a recommendation that a vaccine be prepared from the aspirated secretion.

Inspection and physical examination of the chest revealed rapid and shallow breathing which became more marked upon the slightest exertion. There appeared to be a slight cyanosis of the lips with some retraction of the epigastrium. The appearance of the chest suggested a severe emphysematous condition. The percussion note was hyperresonant over the entire chest and the diaphragmatic excursion was unobtainable. Upon auscultation the breath and heart sounds were feeble and partially obscured by the harsh rhonchi which were present. Radiographic study of the chest showed the lung fields to be darker than normal. The intercostal spaces were widened and both diaphragms greatly depressed.

Bronchoscopic examination revealed a large pedunculated, freely movable tumor mass in the lower one third of the trachea arising from the posterior wall. Because of its changing position during expiration, the tumor mass almost completely obstructed the egress of air. It was a beautiful demonstration of an expiratory check valve.

The base of the tumor mass was quickly grasped with cutting forceps and a large piece was removed. Owing to extreme difficulty in breathing during the endoscopy, no further attempt at removal was made. Within several hours following the operation there was noted a marked improvement in breathing and the patient had lost the expression of fear which is so commonly seen in patients who are fearful of sudden asphyxia. The histological report of the removed tissue was an epidermoid carcinoma, grade 2. Six days later the patient underwent another bronchoscopic procedure at which time the remaining tumor mass was removed and the base destroyed by fulguration. She was advised to undergo a course of deep x-ray therapy and on June 15 the first treatment was given. Using a modified Coutard technique she was given a total of 7500 r. units.

The patient was closely followed and at varying intervals was admitted to the hospital for bronchoscopic examination. Except for a small linear scar in the posterior wall of the trachea at the former site of the tumor mass, there was no further evidence of pathology. Since her discharge from the hospital in June 1942 the patient has enjoyed excellent health and for the past two years she has been working as a saleslady in a department store.

Comment. It is difficult to evaluate whether the deep x-ray therapy which followed the removal of the tumor mass was responsible for the nonrecurrence of the cancer or whether the tumor was entirely removed by bronchoscopic procedure. During the past five years the author has seen two cases of laryngeal carcinoma in which the diagnosis was made by biopsy and later, when the involved cord was examined after removal by laryngofissure, no evidence of carcinoma was found.

It is not the intention of the author to justify the mistaken diagnosis. However, in view of the mode of onset, the presenting symptoms, the x-ray and physical findings, the diagnosis of bronchial asthma seemed a reasonable one. If every practitioner of medicine, particularly the allergist, would come to realize that "all that wheezes is not asthma" and that bronchoscopic examination should be first carried out on all patients suspected of this condition, perhaps a great many more tracheal lesions would be discovered. During the past year there have been several reported cases of sudden death in adults undergoing treatment for bronchial asthma. May not some of these patients, especially those in the cancer age group, have died of asphyxia due to a complete tracheal obstruction from a tumor mass?

It is very puzzling to the author as to why carcinoma of the trachea is rare and yet bronchogenic carcinoma is responsible for ten per cent of all cancer deaths. The histology, except for the membranous portion of the bronchial tract, is the same throughout. Is it because of chronic irritation due to the fact that the dependent portions of the bronchial tree are constantly being invaded by infected drainage material from the upper respiratory tract? Another fact worthy of speculation is that during the past ten years, during which period the incidence of bronchial carcinoma has been high, we have also had the greatest number of automobiles. May not the chronic bronchial irritation due to the daily inhalation of air heavily saturated with carbon particles from the exhausts, be an important carcinogenic factor in explaining this difference? Experimental work carried out with laboratory animals has definitely shown that, when solutions containing carbon particles are sprayed into the nasal chambers, the greater portion of the sprayed material is aspirated into the bronchial tract.

SUMMARY

A case of primary carcinoma of the trachea is presented in which a mistaken diagnosis of bronchial asthma was made. The pedunculated tumor mass located in the lower one third of the trachea caused an expiratory obstruction to the egress of air. This expiratory check valve resulted in bilateral emphysema and the symptoms and physical findings which followed were not unlike those commonly found in bronchial asthma. The knowledge gained from this case clearly proves the often repeated statement of Chevalier Jackson that: "all that wheezes is not asthma."

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LXV

BRONCHOGRAPHIC STUDY OF 555 CASES WITH SPECIAL REFERENCE TO BRONCHIECTASIS

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The development of bronchography is of recent origin. During the early phase, workers in this field were concerned chiefly with securing nontoxic radiopaque substances which could be safely introduced into the tracheobronchial tree for diagnostic purposes. In this period, it was not always thought necessary to obtain complete filling of all bronchopulmonary segments in cases of bronchiectasis. Since the perfection of thoracic surgical technique, it has been found more and more imperative to fill all bronchopulmonary segments to determine the entire extent of any pathologic process such as bronchiectasis in order to make certain whether or not such a lesion can be eradicated completely by surgical procedure.

In 1905 Jackson[1] first used a radiopaque substance to outline the tracheobronchial tree. Jackson used bismuth oxide powder and insufflated this material through a bronchoscope. In 1922, Sicard and Forestier[2] advocated the use of lipiodol as a contrast medium to visualize the bronchial tree. Since that time, different preparations of iodized oil have been produced and used in bronchography.

In the past, many different techniques have been used to introduce the iodized oil into the bronchial tree. These include introduction of the radiopaque oil through a needle through the cricothyroid membrane, by various supraglottic methods, and through a catheter, cannula or bronchoscope directly into the trachea. Of these methods, the intratracheal catheter method has proved to be the most satisfactory in the majority of instances.

Various authors[3-5] have emphasized the importance of securing complete filling of all bronchopulmonary segments in cases of bronchiectasis prior to lobectomy, segmental resection or pneumonectomy. I should like again to point out the necessity of doing this, since it is imperative that all of the involved bronchi be removed at the time of operation if the patient is to be relieved of his symptoms. After lobectomy or segmental resection, the remaining portion of

the lung is subjected to certain physical forces which cause it to expand and to become emphysematous. These same forces are transmitted to the bronchial tree, and if these bronchi are involved with bronchiectasis, they almost certainly will dilate to a greater degree because of their inherent weakness. Thus, if a left lower lobectomy is performed for bronchiectasis and a bronchiectatic lingula is left behind, those bronchi of the lingula that are involved with bronchiectasis almost always will show increased dilatation postoperatively. Similarly, after right lower lobectomy, bronchiectasis of the middle lobe of the right lung sometimes becomes more severe.

This report is based on a series of bronchographic studies of military personnel at Fitzsimons General Hospital, Denver, Colorado, in an 11-month period. This was a selected group of patients, since all of them had passed a physical examination prior to their entrance into military service. Most of them had had roentgenograms of the thorax at that time. Many of these soldiers had gone through rigorous basic training maneuvers and actual combat. At some time or other during their military career symptoms referable to the tracheobronchial tree had developed and were severe enough to necessitate hospitalization, and the patients eventually were sent for complete examination to a center where thoracic diseases were diagnosed and treated. The group included patients from every war theater to which the United States Army was sent.

All patients underwent a complete physical examination, roentgenologic examination of the thorax and examinations of sputum for tubercle bacilli soon after admission to the hospital. If the presence of tuberculosis was suspected, multiple cultures of sputum, gastric and bronchial secretions were made. All such bacteriologic studies gave negative results in the group herein considered. All had undergone bronchoscopic examination previous to the bronchographic studies. It was a rule that all bronchopulmonary segments had to be filled completely before the bronchographic studies were finished. In most of the cases, a single study was made because the technique was one by which bilateral bronchographic studies were performed as one procedure.[6] When adequate filling of the bronchial tree was not accomplished, the studies were repeated until satisfactory filling of all bronchopulmonary segments was obtained.

All patients were asked to take postural drainage exercises just before reporting for bronchographic studies, in order to rid the bronchial tree of secretion. They were given 2 grains (0.13 gm.) of sodium phenobarbital an hour before the studies were made and

Fig. 1.—Severe extensive cystic bronchiectasis.

1/150 grain (0.00043 gm.) of atropine was given 15 minutes before the examination. Cocaine hydrochloride was used as the anesthetic agent. The intratracheal catheter method of introducing the oil was used. Lipiodol and iodochlorol were the two radiopaque oils used in making the studies. Twenty cc. of iodized oil were instilled into the right bronchial tree and the patient was positioned according to a technique described by me. After this, anteroposterior, right lateral and right oblique roentgenograms were made. Roentgenoscopic examination was not attempted prior to making roentgenograms since the delay encountered by this procedure usually allows the opaque material to go too far out into the parenchyma of the lung.

After the study of the right side was completed, the left side was filled by introducing another 20 cc. of iodized oil and the patient was put in position according to the same technique and anteroposterior and left oblique roentgenograms were made.

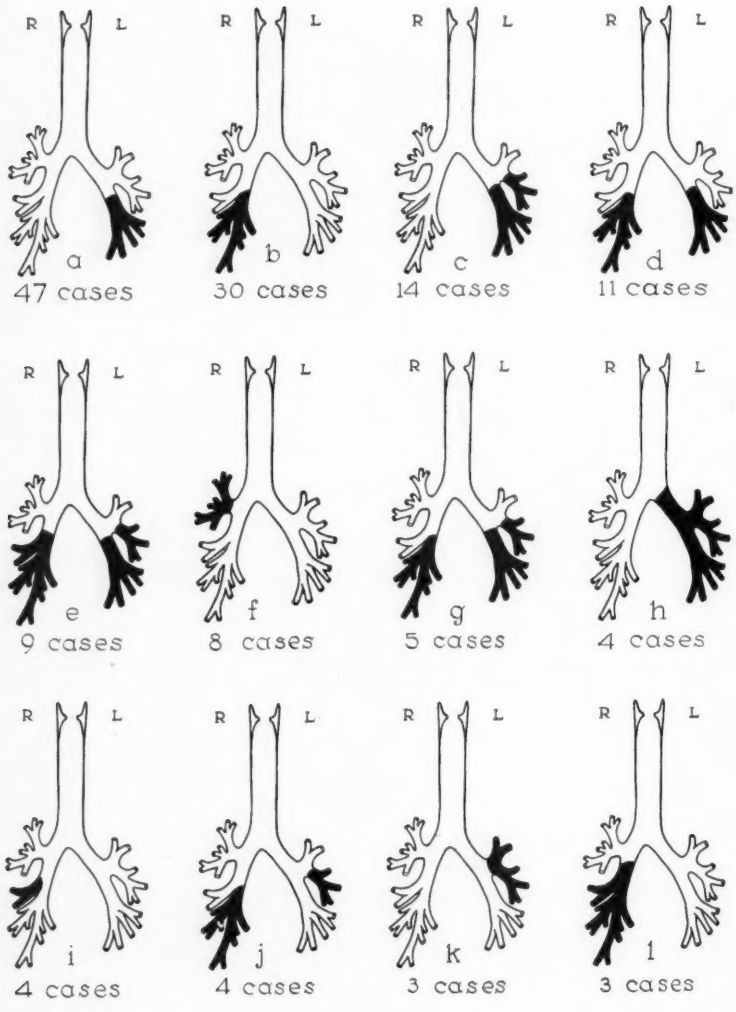

Fig. 2.—Shaded areas represent the lobes involved with bronchiectasis in 158 cases.

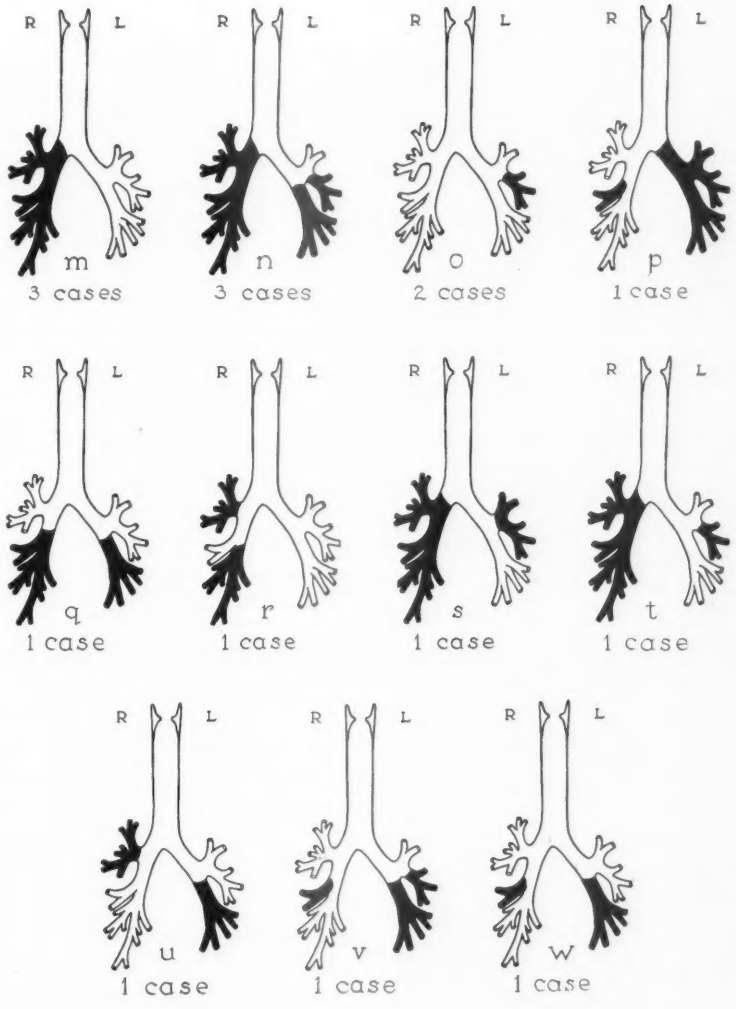

Fig. 3.—Shaded areas represent the lobes involved with bronchiectasis in 158 cases.

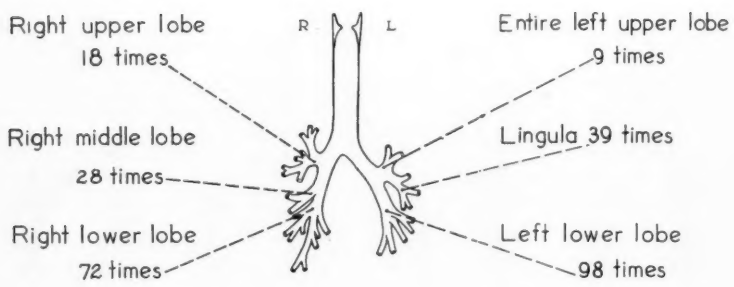

Fig. 4.—Summary of the total number of times each lobe was involved with bronchiectasis.

During a period of 11 months, a total of 555 bilateral bronchographic studies were performed. Of these, 257 showed positive findings. Out of this group, examination revealed evidence of nontuberculous bronchiectasis in 158 cases. The severity of the bronchiectasis varied from minimal to extensive involvement (Fig. 1). The lobar distribution of the bronchiectasis in this group is illustrated in Figs. 2 and 3.

The total number of times that each lobe was involved is illustrated in Fig. 4.

It is of interest to note the relative frequency of involvement of the upper lobes. I feel that active pulmonary tuberculosis was fairly well excluded as an etiologic factor in this group by the negative results of bacteriologic studies in each case. I believe the reason for obtaining such a high incidence of bronchiectasis of the upper lobe was that adequate filling of the bronchi was obtained, the studies being repeated if necessary (Fig. 5*a*).

Four cases of situs inversus viscerum were encountered. All of these patients had bronchiectasis. In these four cases the lower lobe of the left lung was involved with bronchiectasis. The lower lobe of the right lung was involved in three cases and the lingular division of the right upper lobe was involved in two cases. The upper lobe of the left lung was involved in one instance.

It is important to perform bronchographic studies after segmental resection or lobectomy to determine what has happened to the remaining portion of the bronchial tree. We tried to make this a routine procedure at Fitzsimons General Hospital and occasionally

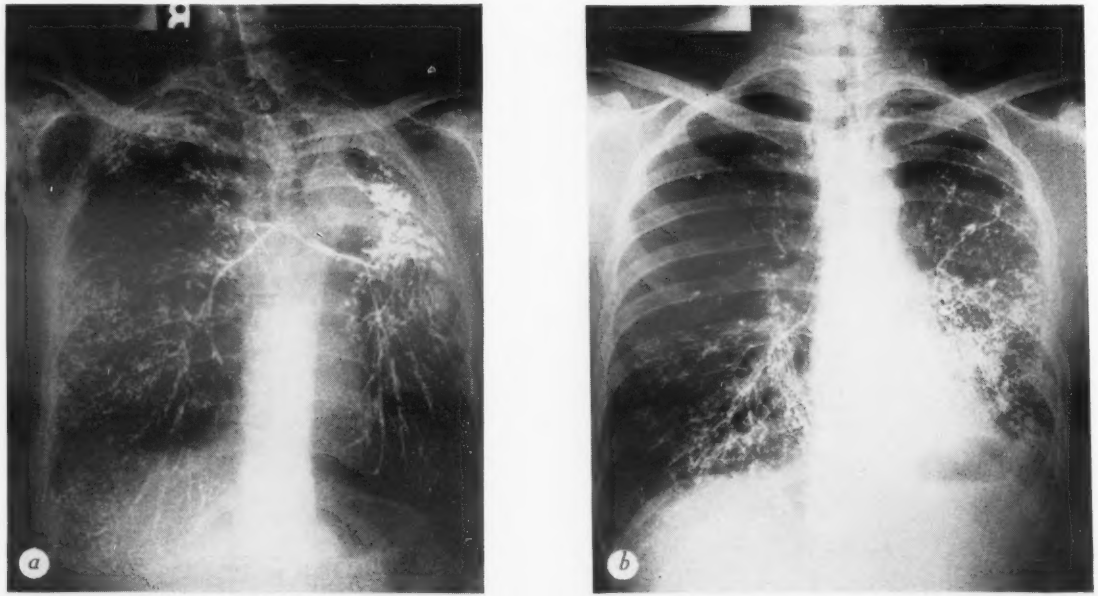

Fig. 5.—*a*. Extensive bronchiectasis in the left upper lobe strongly suggested tuberculosis. A total of 12 negative sputum, bronchial and gastric cultures for tubercle bacilli were made. *b*. Long lingular stump after left lower lobectomy and lingulectomy. The symptoms persisted and resection of this bronchial stump was necessary.

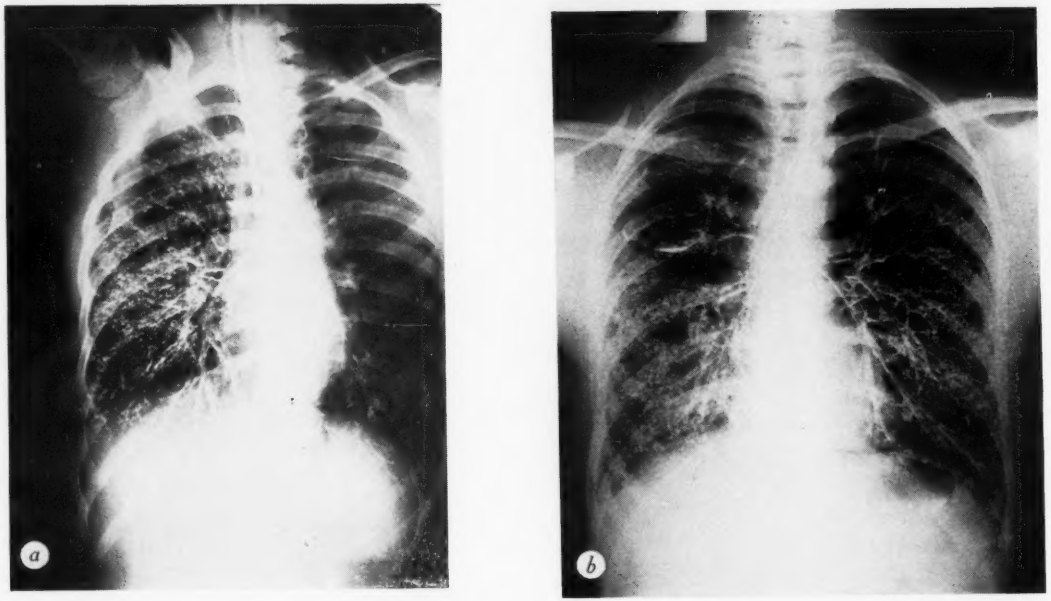

Fig. 6.—*a*. Anomalous distribution of bronchi to the upper lobe of the right lung. The apical division arises from the trachea. *b*. Posttraumatic cystic changes in the upper lobe of the right lung after a penetrating wound from a 31-caliber German machine gun bullet.

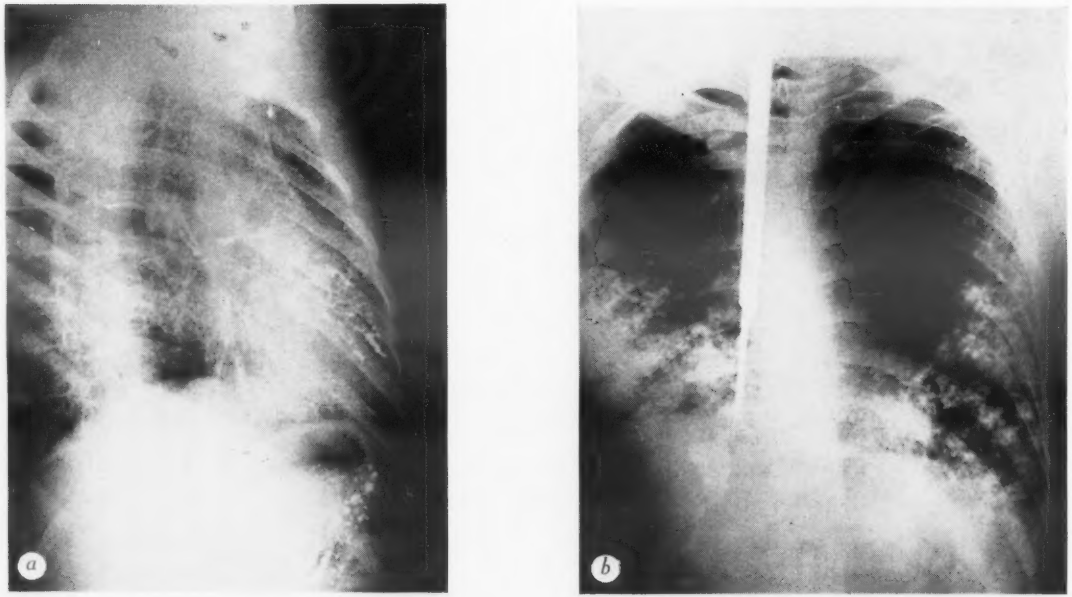

Fig. 7.—*a*. Posttraumatic bronchiectasis of the lingula. *b*. Subphrenic abscess with communication with medial basal bronchus of the lower lobe of the right lung outlined with iodized oil introduced through the bronchoscope.

some of the findings were interesting. In two instances, long lingular bronchial stumps were seen to remain after left lower lobectomy and lingulectomy (Fig. 5*b*). These bronchial stumps retained infected secretions to such an extent that they produced a distressing productive cough in each instance. It was necessary to operate on these patients and remove an additional portion of the lingular bronchus. I have also observed what appeared to be a normal middle lobe bronchial tree become bronchiectatic after right lower lobectomy.

Other positive findings, including congenital anomalies of the bronchial tree, were encountered at the time of the bronchographic studies (Fig. 6*a*). One patient had a cavity due to coccidioidomycosis and the lipiodol entered this cavity during the time of study. Other positive findings included cystic lesions of the lung and abscesses of various portions of the lung.

Many of the patients at the bronchoscopic clinic had had penetrating war wounds of the thorax and of the lung. Of this group, some still showed roentgenologic evidence of cystic lesions of the lung (Fig. 6*b*). Interestingly enough it was possible to fill these lesions with oil. In only one instance was there bronchiectasis which I felt might have been posttraumatic in origin. The patient was a young Air Corps captain who was piloting a B-17 over Berlin when he sustained a penetrating wound of the left side of the thorax from large fragments of a high-explosive shell. A foreign body was removed from the upper lobe by left thoracotomy. After this accident, the patient continued to cough up bloody material, and bronchographic studies showed evidence of cylindrical bronchiectasis of the lingular division of the upper lobe of the left lung (Fig. 7*a*). In some of the other cases in which patients had sustained penetrating wounds of the thorax, evidence of bronchiectasis was found but its distribution in relation to the wounds was such that it was not thought that the bronchiectasis was brought about by the recent trauma. In almost every instance it was felt that bronchiectasis had been present at the time the patient was inducted into the military service. In two instances it was possible to demonstrate subphrenic abscesses which had evacuated themselves through the bronchial tree at the time of bronchoscopy when oil was introduced directly into the involved bronchus by means of a metal bronchial catheter. The routine bronchographic studies in each of these cases failed to demonstrate any such communication (Fig. 7*b*).

MAYO CLINIC.

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LXVI

THE EOSINOPHILIC LUNG

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I have chosen the term "eosinophilic lung," first used by Frimodt-Moller and Barton,[1] to designate a pseudo-tuberculous condition associated with high eosinophilia and better known as Loeffler's syndrome or tropical eosinophilia.

Since Loeffler[2] first drew attention to a condition characterized by changing or transitory pulmonary infiltrations with blood eosinophilia, many similar cases have been reported. Weingarten[3] named the symptom complex tropical eosinophilia, stressing its differences from allergic bronchitis and Loeffler's syndrome. In his cases the predominant feature was massive eosinophilia, responsible for considerable leukocytosis. Several writers mention the striking results obtained in these cases by treatment with the aromatic arsenicals and suggest that without this specific therapy the disease may persist indefinitely.

Lal,[4] who reported 15 cases of tropical eosinophilia, believes that the diagnosis and treatment of "eosinophilic lung" are well known today. He feels, however, that the medical profession is adopting a complacent attitude in accepting this condition as a clinical entity with a specific treatment, the arsenicals.

Kartagener[5] suggested that eosinophilic pulmonary infiltrations be divided into three forms: (1) the Loeffler type, which represents an acute process of short duration; (2) the type described independently by Loehr and Leon-Kindberg et al., in which the symptoms are severe and septic, with lesions which may persist for months; and (3) the type observed by him characterized by chronicity and mildness of symptoms. I agree with Serra[6] in that this should be considered as representing variations of a single entity, rather than separate ones, as he has implied.

Blackfan and Diamond[7] emphasize the occurrence of eosinophilia as the peculiar response of certain individuals to low-grade chronic infection, and Menon[8] states that, from the clinical standpoint, the

theory of an infective organism is compatible with the findings, pointing to infectious mononucleosis as a striking example.

In most reports the syndrome is explained as an allergic response in the lungs to various allergenic substances, including the tubercle bacilli[9] and other bacteria, pollens,[10] Endamoeba histolytica,[11] the larvae of Ascaris lumbricoides,[12] trichinae,[13] and nematodes of cutaneous helminthiasis.[14]

Fleeting bronchopulmonary changes have been described in association with sulfonamide treatment,[15] brucellosis,[16] asthma,[17] measles, whooping cough, tularemia, chickenpox, diphtheria and grippe.[18] In Lal's cases bronchospasm was the most frequent finding. Soderling[19] attributed the lung condition to stagnation of secretions following bronchitis, which four of his five patients had, combined with bronchial spasm and resulting localized areas of atelectasis and emphysema. A transitory atelectasis is generally held responsible for the radiologic opacity. Apparently the bronchi serve as the major shock organ. In Baggenstoss'[20] case, the most significant features aside from the predominance of eosinophilic leukocytes in the pneumonic exudate were the character of the bronchial lesions, the advanced organization of the pneumonic exudate, the granulomatous character of the focal lesions, and the presence of vascular lesions. The bronchial lesions were similar to those seen in bronchial asthma. Hypertrophy of the muscle of the bronchial wall was not pronounced, but the predominantly eosinophilic exudate infiltrating all portions of the wall, the marked hyalinization and thickening of the basement membrane of the epithelium, and the presence of large quantities of mucus in the cells and lumina were all typical of bronchial asthma. According to Baggenstoss, the advanced organization of the pneumonic exudate and the granulomatous character of the focal lesions constitute overwhelming evidence that the pulmonary lesions are not "transitory" in all cases. Von Meyenburg[21] maintains that the infiltrations represent an eosinophilic pneumonitis.

Referring to the syndrome under discussion, Harkaway[22] suggests that these cases may be regarded as formes frustes of diffuse hyperergic vascular disease. He pleads for early diagnosis and individualized therapy directed to the elimination of sources of infection in the hope of reversing the hyperergic response and arresting the disease process.

I am entirely in accord with Lell's[23] statement that any form of pulmonary allergy is primarily a medical problem requiring extensive individual study to determine the underlying cause. However, my

inclination to investigate the bronchospasm and persistent cough so frequently accompanying this symptom complex involved me in a controversy—on one hand, the association of eosinophilia with pulmonary infiltration as recorded by Loeffler in Switzerland, on the other, a more serious condition occurring in the tropics, the Weingarten syndrome. In this connection, Apley and Grant[24] suggest that the differentiation of Loeffler's syndrome and tropical eosinophilia is more apparent than real and that the gradation between the two conditions renders it more profitable to consider them as manifestations of a single disease process.

The blood eosinophilia is the most frequent accidental discovery, while the paroxysmal, dry, hacking cough is the complaint which most frequently brings these patients to the laryngologist.

REPORTS OF CASES

Tropical Eosinophilia (Weingarten's Syndrome).—A mulatto, aged 10, was admitted to the Presbyterian Hospital, San Juan, Puerto Rico, on October 26, 1946, because of a painful swelling of the left hip of about 20 days' duration, accompanied by fever. During the past two months he had had a cough and two asthmatic attacks, the last one about three weeks prior to admission. Before the present illness there had been no complaints referable to the respiratory tract. The general health had been good, with no serious illness except pneumonia at three years of age.

The patient's mother had died of pulmonary tuberculosis. The father had died of rheumatic heart disease. Six siblings were living and well.

The patient was well developed but poorly nourished, lying quietly in bed and apparently in no acute distress. The outer surface of the left hip presented a hard, tender, warm area with no fluctuation and unrelated to any trauma. The white blood count on admission was 26,800, with 63 per cent eosinophiles. X-ray examination showed no evidence of bone disease of the left hip joint. X-ray examination of the chest was reported as follows: "There is a marked enlargement and accentuation of the hilar shadows and pulmonary stroma, with some peribronchial reaction and a few fine areas of mottling in both lungs. The heart is normal in size, shape and position. The picture is suggestive of an eosinophilic infiltration of the lungs. Tuberculosis cannot be entirely ruled out."

Blood, urine and stools were negative for parasites. Sputum and nasal secretions showed no eosinophiles, parasites or tubercle bacilli. The tuberculin test and skin tests with schistosoma, taenia and trichinosis antigen were all negative. Serologic studies for syphilis and the agglutination test for Malta fever were also negative. The sedimentation rate was 34 corrected. Sternal puncture showed an active marrow with a high percentage of eosinophiles. Ten days after the patient's admission the white blood count was 25,450, with 69 per cent eosinophiles.

On November 20, a diagnostic bronchoscopy showed, following the aspiration of abundant white, viscid, frothy secretions, several white patches of exudate scattered throughout the bronchial mucosa, especially in the region of the carina and the main orifices. On removal of portions of the exudate, there was no bleeding and no evidence of inflammatory reaction of the mucosa. Examination of the exudate showed no acid-fast organisms, parasites or fungi. No eosinophiles could be seen. Culture of the aspirated material yielded pure strains of Streptococcus viridans.

Arsenical treatment was instituted, and Carbarsone was given in daily doses of 0.25 gm. for ten days. During this period the temperature remained elevated and the hemograms showed 53 per cent eosinophiles on the fifth day, 63 per cent on the tenth day.

A sinusotomy was carried out to drain both antrums. At this time the hemogram showed 20,600 leukocytes, with 60 per cent eosinophiles. Cytologic examination of material from the sinuses showed no eosinophiles. On culture, Streptococcus viridans and a pneumococcus were found.

Three days after the antrotomies, on December 5, the white blood count had fallen to 13,450 and the eosinophiles to 14 per cent. The next day the white blood count showed a further drop to 9,850.

The patient was discharged on December 26 with a white blood count of 8,500 and 16 per cent eosinophiles. There was practically no change in the appearance of the lung fields, but the findings were negative in two subsequent bronchoscopies.

Loeffler's Syndrome. A white female, aged 9, was first seen in my office because of a persistent dry, hacking cough. For the previous two weeks she had been wakened at night by spasmodic attacks of coughing and wheezing. Several hemograms obtained prior to my consultation showed a varying leukocytosis and mild eosinophilia.

Roentgenologic examinations of the chest had shown varying degrees of pulmonary infiltration. There was no evidence of a toxic state and no elevation of temperature at any time. There was no personal or family history of tuberculosis, asthma or any allergic condition. The patient had been in good health until she suffered an upper respiratory infection of short duration. Arsenicals and the antihistamine preparations had been tried without success.

The patient was admitted to the Presbyterian Hospital for observation and study. The diagnostic work was directed first of all to the exclusion of tuberculosis. Dr. Ortiz, Chief of the Pediatric Service, summarizes his findings as follows: "We have the following evidence against the diagnosis of acid-fast infection: (1) the excellent general condition of the patient, the absence of fever and associated constitutional disturbances; (2) the history of paroxysmal cough and dyspnea with grossly audible wheezes; (3) negative tuberculin tests; (4) blood eosinophilia, absence of acid-fast organisms but many eosinophiles in the sputum; (5) no elevation of sedimentation rate." He concludes by saying that in his opinion the disturbance is allergic in nature. "It could be called Loeffler's syndrome or recurrent nocturnal asthma, with pulmonary infiltrations, however."

Ortiz further stated, "The coexistence of an infantile type of tuberculosis cannot be ruled out with mathematical certainty, but it can be ruled out as a significant factor on clinical grounds, including negative laboratory evidence."

Bronchoscopy disclosed a hemorrhagic, red, edematous mucous membrane, reducing the caliber of the lumina of the trachea and both main bronchi, and the presence of thick, tenacious secretions. The secretions obtained with the bronchoscope showed no acid-fast organisms, parasites or fungi, but there were many eosinophiles. Culture of the aspirated material showed Streptococcus viridans and hemolytic Staphylococcus aureus.

Roentgenologic examination of the paranasal sinuses had shown a chronic hyperplasia of the left maxillary sinus. An antrotomy was performed, and the antral secretions yielded a pure culture of Staphylococcus aureus hemolyticus. Penicillin was given preoperatively and postoperatively.

At the time of her discharge from the hospital, the patient's white blood count was 5,750, with 16 per cent eosinophiles. Since then she has been receiving weekly injections of an autogenous vaccine derived from the antral and bronchial material and consisting

of Streptococcus viridans and hemolytic Staphylococcus aureus. At the time of this writing, her condition is excellent. The cough has disappeared, the lung fields are clear, and the hemogram shows 7 per cent eosinophiles.

Comment. On the basis of observations in two cases subjected to bronchoscopy, it would appear that there is no constant relationship between the tissue and blood eosinophilia, this apparently being controlled by the virulence of the infection and the response of the body. The total eosinophile count seems to reflect fairly accurately the degree of individual sensitivity. The role of the presence or absence of eosinophiles in the bronchial secretions cannot be evaluated. However in these two and eight other cases which I have seen the greater number of eosinophiles is found in the bronchial secretions of patients with Loeffler's syndrome as against the relatively fewer or complete absence of eosinophiles in the secretions of those patients with tropical eosinophilia.

It is questionable whether an etiological grouping of the allergic pulmonary syndrome can safely be made on the basis of the symptoms and the lesions.[25]

Nevertheless, the response of the lung lesions and the blood eosinophilia to treatment emphasizes the definite relationship between these and the offending organism; thus, a diligent search for foci of infection in the oral cavity, the nasopharynx and its accessory sinuses, and bronchoscopic study might help to clarify this interesting syndrome and further enhance our position in the field of modern medicine.

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LXVII

LIPOMA OF THE BRONCHUS

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Tumors arising from the wall of the bronchi, while they may be histologically benign, can be disturbing to body functions and sometimes destructive to the life of the host. A benign tumor, by general acceptance, is one that does not metastasize and does not invade surrounding tissue.

Before endoscopy made possible the inspection of the bronchial tree, these benign tumors were on occasion found at autopsy. With the development of bronchoscopy, such tumors were recognized as being part of the clinical picture seen in bronchiectasis, bronchostenosis, atelectasis and lung suppuration. Any tumor occluding a bronchus can be the cause of these changes in the lung.

A benign tumor, in this instance a lipoma, which would be a harmless growth if located on an exposed surface of the body and would not call for treatment, can be the cause of a disastrous situation when located in the bronchial tree. With the growth of the tumor comes the blocking of the airway, insufficient aeration, stagnation of secretions and infection of that section of the lung. Such changes are only in part controlled by chemotherapeutic measures and the victim goes on to chronic disability or death.

A lipoma is a firm, elastic, spherical tumor, circumscribed but not encapsulated. Lipomas constitute about five per cent of all benign tumors but are one of the less common tumors of the bronchus. Several clinical and anatomical types of lipoma are recognized—subcutaneous, intramuscular, synovial, intracranial and visceral. Of these, the subcutaneous lipomas are the most common. A congenital tissue predisposition seems to be an essential factor in the origin of most lipomas.

There is a striking tendency for these tumors to occur in certain locations. In the male, they are common about the region of the neck; in the female, they are frequently encountered in the breast. Such tumors are apt to be located in the subcutaneous fat of the back and buttocks and are frequently multiple and ordinarily remain

benign. I have not seen such multiple lipomas in my cases of bronchial lipoma. Cases have been reported in which as many as two to three hundred such small subcutaneous nodules have been found. They tend to grow very slowly and reach a phase of equilibrium with no further increase in size, sometimes for many years. They vary greatly in size from small ill-defined nodules one centimeter or so in diameter up to huge tumor masses which hang as pedunculated tumors from the skin, particularly on the back. Occasionally such tumors may be of serious importance because of their location and in particular is this true of those instances of mediastinal involvement where, from pressure upon the nerves and great vessels, even a fatal outcome may ensue. They have been found in the pharynx, larynx and esophagus and in these locations can cause alarming symptoms from interference with the food passage or airway.

Connective tissue, cartilage and bone all differentiate from that type of mesoderm known as mesenchyme. Mesenchyme arises primarily from the primitive streak and secondarily from mesodermal segments and the lateral somatic and splanchnic layers. It is seen as a spongy network composed of branching cells whose processes touch rather than actually anastomose; between the cells occur open spaces which are filled with a ground substance of coagulable fluid. In early embryos, the mesenchyme acts as a packing material between the external and internal epithelia but it soon differentiates into various tissues and organs. Of these the inert supporting tissues are peculiar in that a ground substance or matrix appears which usually becomes bulkier than the cellular elements themselves. The matrix may be fibrous, cartilaginous or bony in nature; in each instance it is a question whether the matrix originates within the substance of the mesenchymal cells or is merely organized and laid down in the spaces between them.

In the development of the embryo, the fat cells are derived directly by differentiation from the original mesenchyme and therefore represent a variety of connective tissue. In the course of the differentiation of these cells, they appear in islands as the so-called fetal fat cells, which are large ovoid cells with rather large nuclei and a finely vacuolated cytoplasm. These do not look unlike aggregations of large mononuclear phagocytic cells except that the nucleus is likely to be more characteristic. As the body develops further, these cells go through a transitional stage, developing into the adult type of fat cell. In this process, the neutral fat accumulates within the cytoplasm of the cell to such an extent that the nucleus is com-

pressed and pushed to one side to produce the characteristic "signet ring" appearance. Both from the fetal and the adult types of cell, tumors may arise. These tumors are more readily diagnosed grossly than microscopically because histologically the tumor is found to be composed wholly of normal appearing cells. It is only the separation of these cells from the surrounding adipose tissue by a thin, often almost membranous connective tissue capsule that identifies and distinguishes them from normal supportive body fat.

Certain of the mesenchymal cells give rise not to fibroblasts but to lipoblasts which are the forerunners of fat cells. They elaborate within their cytoplasm droplets of fat which increase in size and become confluent. When at last a huge droplet fills the cell, the nucleus and cytoplasm are pressed to the periphery. Fat cells arise in close association with developing blood vessels in areolar tissue and appear during the fourth month; the primitive clusters are foci for the later lobules. It is not possible to determine whether the lipomas in adults are derived from normal adult fat tissue, from remnants of fetal fat or from perivascular undifferentiated mesenchymal cells.

At various points in the fetus, there are groups of distinctive granular lipoblasts which are set apart as so-called adipose glands. Fat cells derived from them are peculiar in containing multiple fat droplets which for a time do not coalesce, but in early infancy they become indistinguishable from the ordinary type. In the cat, this specific tissue continues until maturity and in rodents it constitutes the permanent hibernating gland. According to one theory it is a cell set apart in early life for the producing of fat, a so-called primitive fat organ, being found at various points in the form of small groups of stellate or spindle cells richly permeated by capillary vessels. The other theory accounts for the origin of the fat cell on the basis of metaplasia of the fibroblast with the accumulation of fat within its body. The fat cell when it fails to take on the specialized function of the fat cell, it is assumed, follows its natural inclination and develops into connective tissue.

The presence of fat in many benign tumors of the bronchus has been the subject of an interesting study by Watts, Clagett and McDonald[1] at the Mayo Clinic. Their interest was aroused by the reports of the finding of fat in so many tumors of the lung. In the reported cases, fat has been found in more than half of the chondromas, all of the fibrolipomas and in many of the adenomas of the bronchus. It had been suggested that this fat could be an extension of the fat that is found in the submucosa of the bronchi. It might

be from a metaplasia of the connective tissue or it could be from embryonic tissue destined to become fat that had been carried from the mediastinum by the outgrowth of nerves, blood vessels or bronchi. Flint's[2] work on the embryology of the lung indicated that some tumors of the lung might result from the failure of the embryonic bronchial buds to develop into normal structures. Because the bronchial buds originally consist of mesoderm and entoderm, such a theory would explain the occurrence of certain growths in which both mesothelial and entodermal elements are present. If this theory is acceptable, then the fat found in tumors of the lung would arise from embryonic mesoderm.

In this study at the Mayo Clinic, the investigators posed these questions: (1) Is fat a normal component of the bronchus? (2) If it is, in what portion of the bronchial wall does fat occur? (3) In which of the bronchi is fat present?

The material used in this study was the bronchi and parenchyma of 15 lungs, surgically removed. Other autopsy material was used to round out the study. Their findings in substance were that, in the large bronchi, fat was always found in the fibrous tissue external to the cartilage plates. A lesser amount of fat was found in the submucosa between the cartilage and the muscular layer. In the smaller bronchi, fat was present in the walls in diminishing amounts until the cartilage disappeared. It was not possible to find fat in any part of the bronchial tree that did not contain cartilage. As was true in the larger bronchi, fat was found in abundance in the fibrous tissue external to the cartilage.

The conclusions arrived at from this study were that fat is a normal constituent of the bronchial wall and that it is unnecessary to provide a theory, based on development or metaplasia, to explain the origin of a lipoma of the bronchus. It is probable that these tumors arise from adipose tissue normally present in the submucosa. These conclusions give an explanation for the finding of small amounts of fat in bronchial adenomas.

The clinical picture of a benign tumor of the bronchus will be that resulting from mechanical obstruction to the airway. Such an obstruction will disturb the necessary exchange of air and will interfere with the drainage of the section of lung which is obstructed. The picture will differ depending on whether a stem bronchus is obstructed or whether the growth is in a minor bronchus. Since the common site for a lipoma of the bronchus is in one of the stem bronchi, the bronchial obstruction will result in the impairment of

an entire lung. A lipoma is a slow growing tumor, benign in nature, which does not break down, and so the progress of signs and symptoms will be over a long period of time. As a rule, the first symptoms will be those resulting from bronchial obstruction. Such an obstruction to the exchange of air and proper drainage can cause a local pneumonitis, lung abscess or other forms of lung suppuration. Wheezing will be present if the tumor allows air to pass to and fro in the bronchus. Hemoptysis will not be observed as a rule since there is no breaking down of tissue. Blood-tinged sputum may be observed as a result of secondary changes in the parenchyma of the lung from stagnation of the secretion and associated infection. The x-ray findings will be typical of an atelectasis of sections of the lung but this is true only when the growth becomes obstructive. As endoscopists, we recognize the indication for a bronchoscopic examination when presented with x-ray evidence of atelectasis, interference with proper exchange of air or faulty drainage of parts of the lung. Atelectasis of one lobe is sufficient to cause anoxemia of the aortic blood and this is plainly visible as cyanosis in the fingers, lips and ears.

The following is a description of a typical case of a benign tumor of the bronchus.

REPORT OF A CASE

A white male, aged 61, entered the hospital November 5, 1946, with the complaint of chills, fever, and pain in the chest of three days' duration.

He gave a history of two similar attacks, the first in 1944, the second in 1945. At the time of the first attack he was found to have a marked wheezing along with a rise of temperature and chills. The clinical diagnosis was an unresolved virus pneumonia. This diagnosis was based on x-ray findings of an extensive area of density involving most of the left lung. He made what was thought to be a good recovery from this illness. His second attack nine months later was similar to the first.

Examination on admission to the hospital in November, at the time of the third attack, showed a well-nourished man, acutely ill with elevated temperature. Wheezing was not observed. His white blood count was 16,000. The sedimentation rate was 82 mm. in the first hour. The x-ray film was reported to show the heart and the mediastinum displaced to the left. The right lung was clear.

There was complete opacity of the entire left lung field. The films were interpreted as indicating a partial atelectasis of the left lung.

Ten days after his admission, the Bronchoscopic Service was requested to see the patient and the following day a direct examination of the bronchial tree was carried out. Bronchoscopic examination showed the left stem bronchus occluded by a round firm mass with a smooth surface and attached by a broad base at a point 3 cm. from the bifurcation. This globular tumor was removed with some difficulty due to the smooth surface and the almost cartilaginous hardness. The bronchi distal to the tumor contained a moderate amount of thin purulent secretion. No other changes were noted in the bronchial tree. The tumor was composed of adipose tissue with a moderate amount of fibrous tissue between the fat cells. It was described as a lipoma.

The day following the removal of the tumor, the man complained of extreme distress on breathing. On palpation, air was found in the soft tissues of the cervical region and over the upper part of the chest. An x-ray film on this day was reported to show the mediastinal shadow in the midline but considerably wider than is usually seen. A subcutaneous emphysema was noted in the superior thoracic and lower cervical regions, bilaterally. The x-ray findings were thought to be consistent with a mediastinal emphysema with an early mediastinitis. The man appeared acutely ill at this time. After four days of medication with sulfonamides and penicillin, the temperature returned to normal, the crepitation in the skin disappeared and a film of the chest showed that the atelectasis of the left lung had cleared. The mediastinum was still displaced to the left and this was now thought to be due to mediastinal adhesions.

Comment. In review it was believed that this man had a slowly growing lipoma of the left stem bronchus which partially occluded the lumen. His first attack in 1944 was probably a localized pneumonitis from obstruction that was not complete and so respiration produced a wheezing sound. At this time, chemotherapy controlled the infection, better aeration was established and his condition improved. The second attack in 1945 was a repetition of the first.

At the time of this third attack in November 1946, the growth had completely blocked the lumen of the bronchus and he again had a localized pneumonitis. No wheezing was observed since no air passed the obstruction which at this time was complete.

At the time of the removal of the tumor, it is probable that the wall of the bronchus was perforated allowing air to escape into the mediastinum and along the fascial planes to the neck and chest. This must have been a small leak since the air was absorbed after three days.

The mediastinum is a space containing solid structures that are of prime interest to the laryngologist and the endoscopist. The anatomical divisions used in describing the mediastinum are so confusing as to terrify the student and prevent his forming a clear mental picture of this important space and its contents. It is sufficient to think and speak of the mediastinum as a space in the middle of the thorax, bounded laterally by the pleurae, anteriorly by the sternum, posteriorly by the dorsal vertebrae and inferiorly by the diaphragm. We may further divide this space into the anterior and posterior mediastinum, the dividing line being the posterior wall of the trachea.

Emphysema of the soft tissues of the neck and chest is a common finding following trauma. It is a frequent finding in injuries from automobile accidents and not uncommon following tracheotomy and instrumentation of the air and food passages. In these cases air has passed into the mediastinum and has leaked out through the superior aperture of the mediastinum to the soft tissues.

In most cases of mediastinal emphysema there is a leakage of air out through the superior mediastinum into the tissue planes of the neck spaces. In the occasional case in which the air in the mediastinum does not escape into the soft tissues, death is likely to occur from compression of the large vessels in the mediastinum. Marked subcutaneous emphysema is not in itself an indication for other than conservative treatment. In fact those cases of subcutaneous emphysema are, as a rule, less serious than those which show no air in the soft tissues. With increasing respiratory distress, aspiration of the air from the mediastinum can be carried out. A No. 20 gauge needle, attached to a 50 cc. syringe is inserted into the third interspace, 1 cm. to the left of the sternal border and directed medially, parallel to the internal surface of the sternum. Twenty to forty cc. of air may be aspirated with safety and immediate relief of the pressure.

A benign tumor of the bronchus will remain hidden until revealed by endoscopic examination. Such a tumor will be a possibility in all cases of persistent cough, wheezing, lung suppuration, bronchi-

ectasis and x-ray evidence of atelectasis. Not all of these signs and symptoms will be due to bronchial obstruction but, with a routine endoscopic examination of the bronchial tree in all cases of bronchostenosis, wheezing and localized atelectasis, these tumors will be recognized and definitive treatment carried out.

750 Main Street.

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1. Watts, C. F., Claggett, O. T., and McDonald, J. R.: Lipoma of Bronchus, J. Thoracic Surg. 15:132-144 (April) 1946.

2. Flint, J. M.: Am. J. Anat. 6:1, 1906

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, February 3, 1947

THE PRESIDENT, DR. FRANCIS L. LEDERER, IN THE CHAIR

Obliteration of the Endolymphatic Sac and Duct In the Monkey

JOHN R. LINDSAY, M.D.

(Abstract)

Experimental information regarding the function of the endolymphatic duct and sac has been meager. The experiments performed by Guild on the guinea pig in which India ink particles injected into the cochlear duct were eventually recovered in the endolymphatic sac indicated a flow of endolymph in that direction. The theory was proposed by Hallpike and Cairns that a diminution of the normal perisaccular vascular supply was the cause for development of idiopathic hydrops of the labyrinth in Ménière's disease.

The endolymphatic sac and duct were destroyed in varying degrees in seven monkey ears by means of a fine drill. The animals were sacrificed in periods up to three and one-half months. In four of these ears the endolymphatic sac was completely destroyed. The central dilated portion of the duct which is thought to be concerned with resorption of endolymph was also completely destroyed. Only the primary dilatation of the duct at the entrance of the cochlear and saccular ducts and a variable portion of the narrow part of the duct leading away from the primary dilatation were left undamaged. The destroyed parts were replaced by new bone. Histologic examination showed the endolymphatic system in the cochlea, vestibule and semicircular canals to be entirely normal.

Since no alteration in the volume of endolymph had been produced in periods up to three and one-half months by obliteration of

these portions of the endolymphatic duct system, it is evident that in the monkey the maintenance of the normal quantity of endolymph is not dependent upon this structure alone. The explanation of hydrops of the labyrinth on the basis of interference with the endolymphatic duct appears to be disproven.

DISCUSSION

DR. BARRY J. ANSON: From Dr. Lindsay's series of seven operated ears, it would appear that in at least two all portions of the endolymphatic duct and sac had been successfully destroyed without concomitant disturbing damage to other parts of the otic duct system.

The portions which were experimentally destroyed are, as Dr. Lindsay has pointed out, those which we have come to regard as chiefly functional in maintaining a normal quantity of endolymph within the system of continuous, labyrinthic, epithelial tubes. By the same token, we would have expected to learn that, as a result of ablation, hydrops of the labyrinth would occur.

Dr. Guild's study was experimental, his conclusions based upon observation that injected dyes were deposited (a suitable time after their introduction into the otic system) in the walls of the specialized portions of the endolymphatic duct. Our conclusions were based wholly upon microscopic examination of sectioned material; we believed that the histologic structure of the duct and sac met the requirements of a resorptive mechanism: the epithelial walls were plicate, the subjacent tissue vascular. Recently this opinion has been strengthened by reconstructions, prepared by Dr. T. H. Bast, of the endolymphatic duct and sac and of their blood vessels in human fetuses. The endolymphatic sac was shown to be relatively very large, its walls, proximally, folded to a striking degree; plexiform veins were traced to meningeal and associated veins.

Now I am perplexed, being beset by seemingly contradictory evidence. Several feeble ways of escape from this dilemma may be offered by the following possibilities:

1. That there may be other, ordinarily auxiliary, ways of passage for the endolymph (by way of the dilated proximal portion of the duct or by way of the cochlear aqueduct, across periotic tissue into haversian vessels).

2. That hydrops is an extreme condition, dependent upon disturbance more profound and extensive than that produced by Dr. Lindsay's experimental ablation.

3. That the normal "reclamation" of endolymph is slower than we have supposed—and capable of being managed, for a time, by a process of transudation across other parts of the labyrinthic system into surrounding spaces and tissues.

Were any one of these possibilities to find support in later studies, our concept would merely need modification. But, for the present, I wish to record my admiration of Dr. Lindsay's investigation and my recognition of the great value of such experimental methods. They come closest to permitting actual observation of the mechanisms in action.

DR. JOHN R. LINDSAY (closing): Dr. Shapiro asked whether studies on hearing were made. There were none, and no estimation could be made relative to hearing. These were all monkey experiments and conditioning to sound was not attempted. No nystagmus was seen after operation. There was no deviation of the eyes on pressure over the ear after operation, such as may be produced after fenestration. On the following morning the animals were running around as actively as before operation, and no nystagmus or disturbance of equilibrium was observed.

I want to thank Dr. Anson for his discussion. While the information obtained by the experiments is limited, it is of practical interest from the standpoint of pathogenesis of labyrinthine hydrops.

Changing Concepts of Therapy of Chronic Sinusitis

LAWRENCE J. LAWSON, M.D.

(This paper appeared in full on page 416 of the June 1947 issue.)

DISCUSSION

DR. O. E. VAN ALYEA: Dr. Lawson has given us an admirable presentation of many facts concerning the nose and sinuses as we know them today. It is a broad subject that cannot be covered in a single paper. However, he has given his solution of many important problems encountered daily in rhinologic practice. The main points of his paper might be summed up in the following:

1. A rhinologist must, primarily, be a good physician. He should have a working knowledge of endocrinology and allergy, and should manage the desensitization of his allergic patients personally.

2. He should not overlook the possibility of functional disorders being a cause of symptoms referred to the nose. He should treat each patient as an individual case, rather than as one of a group who complain of symptoms thought to be due to sinus disorders.

3. The management of chronic sinusitis depends upon the establishment of adequate drainage facilities.

4. Functioning structures in the nose and sinuses should not be subjected to mutilating and destructive surgical procedures; this includes the lining membranes of the sinuses.

I agree absolutely with him on all these points and might add that rhinology practiced properly calls for adherence to all the principles contained therein.

Dr. Lawson mentions the middle meatal approach to the maxillary sinus, but is not convinced that it is the method of choice. I assume that he has not been using this method very long, for I have noticed that the longer one uses this approach, the more satisfactory he finds it and the less likely he is to resort to bony puncture of the inferior meatal wall. I find that I use an available opening in two thirds of the cases and in the remaining one third I puncture through the membranous wall of the middle meatus. So far, no untoward effects have been associated with this technique, and patients as a whole are pleased that they do not have to submit to a crushing, forceful, head-jarring procedure.

It is true that manipulations about the ostium injure the ciliated mucosa, but it recovers rapidly and these sinuses improve as quickly as those irrigated through some other area. The assumption is that the procedure should be carried out gently.

Cases of long-standing sinusitis fail to heal because, as Dr. Lawson says, drainage is inadequate. A large number of these will respond to conservative measures. The infection will clear up when drainage facilities reach the point where secretions leave the sinus as rapidly as they form. In certain cases, the ostium is so constricted by thickened tissue that another opening is required. It has been noted that following intranasal antrostomy the lining membrane of the sinus has a tendency to return to a normal state. This fact, plus the success of conservative measures, has practically eliminated the need of the radical procedures and, as Dr. Lawson says, the Caldwell-Luc approach is reserved for those conditions requiring direct

inspection in treatment of new growths, cysts, osteomyelitis and fractures of the sinus walls.

There is a strong plea in Dr. Lawson's paper for a better understanding of nasal and sinus conditions and for their management by nonsurgical measures when, after careful study, these are shown to be properly indicated. I think we may well apply to this paper the remark made by our good friend William McNally, of Montreal, who feels that, regarding the nose and sinuses, rhinologists should "try to understand, but what they don't understand they shouldn't destroy."

DR. JOHN J. BALLENGER: I would like to ask if Dr. Lawson knows of any effect that dust, or any other type of allergen, would have on ciliary activity.

DR. ROBERT LEWY: I should like Dr. Lawson's opinion as to how frequently the maxillary sinus should be irrigated if pus is found in it.

DR. GEORGE SHAMBAUGH: I think Dr. Lawson's paper is very timely because there is a lot of confusion among doctors in general, even among laryngologists, as to what sinus infection is. In reports in the literature we find descriptions of sinusitis when there were no facts to prove it. We know from our observations that most patients who say they have a "sinus" do not have sinusitis. I think we should clearly differentiate rhinitis with reaction in the nose but without retained secretion, from sinusitis with retained secretion; in the latter instance the diagnosis of sinusitis is in order. I think most cases of intumescent rhinitis, hypertrophic rhinitis and catarrhal rhinitis are allergic.

I have been one of Hansel's students for years. He has contributed the minute dosage therapy for allergy, especially dust allergy; as time goes on the dosage has become smaller. He has now, and we have also, cut the doses in some cases to one to one-billion (1-1,000,000,000) dilution, which sounds extraordinary. There are actually patients who are so sensitive that they require dilution as great as that. Dr. Hansel no longer tries to build patients up to the highest dosage; he finds the optimum dose and stops there. If there is a clinical response, he stops at that dosage and carries the patient on a maintenance dose. This small dosage therapy, which is not accepted by most allergists, I think will prove to be one of his greatest contributions.

DR. FRANCIS L. LEDERER: I have been worried about the terminology for years. We speak of symptoms to describe an entity; then we use basic etiology; and we wonder if we are talking about basic pathology or histology. I am referring to the term intumescent —perhaps Dr. Shambaugh is right in thinking that in the majority of cases these are allergic processes.

DR. G. H. MUNDT: I have lived through the period of the conception of the cause of sinus disease, as have many men here. I believe that pneumatization of the sinuses tells a great deal about the prognosis. Some of us think that all patients who come with a complaint of drip in the throat have sinus infection. If one rules out a definite mucous membrane or sinus defect, he can probably reassure these people that they do not have sinus infection. I wish I might be as certain as some physicians are when to wash through the normal ostium and when to go in under the inferior turbinate. Some go through the normal ostium, others through the inferior meatus, and apparently the results are about the same. It has been my experience, however, that if a sinus is much infected, the results are better by flushing through the inferior meatus. Individuals who have a definite osteomyelitis or nonsecreting cyst or polyp in the maxillary sinus might just as well have a Caldwell-Luc procedure at the start, rather than waiting for months; I doubt that they will get well without this simple procedure. Some of the most successful cases I have had are those in which this was done.

I believe that in the last five years I have seen more infection in the maxillary sinus following extraction of teeth than in previous years. We should try to teach men who are doing exodontia that these patients should be taken care of at once, and that an effort should be made to close the opening into the maxillary sinus as soon as the tooth is extracted; the results of neglect are disastrous in some cases. Occasionally one will close under good management without the Caldwell-Luc procedure, but some weeks after the sinus may be full of pus. I am satisfied that many of them have an osteomyelitis that will not clear up under local treatment.

DR. LAWRENCE J. LAWSON (closing): Dr. Van Alyea has had a great deal of experience in teaching this subject and has done much work in the laboratory. I appreciate his contribution.

Dr. Lewy asked about the frequency of irrigation. I think that we have all had experience with patients who have interrupted treatment to go away. When they report back the antrum has cleared. We should not start irrigations until we are sure the infection will not

clear spontaneously. Irrigations every third day give excellent therapeutic response.

I was glad to hear Dr. Shambaugh say that we should not give high dosage in allergic rhinitis, but the smallest maximum dose that will maintain good results. One controversial subject was mentioned —whether the apparent hypertrophy we sometimes see is actual tissue increase or just an intumescence. I think that subject requires further study.

Books Received

Histopathology of the Ear, Nose and Throat.

By Andrew A. Eggston, B.S., M.D., Director of Laboratories, Manhattan Eye, Ear and Throat Hospital; Clinical Professor of Pathology (Post-Graduate Division), New York University Medical College; Director of Laboratories of The Mount Vernon Hospital; Pathologist, Harlem Eye and Ear Hospital; formerly Associate in Pathology, Vanderbilt University Medical School, and College of Physicians and Surgeons, Columbia University; and Dorothy Wolff, A.B., M.A., Ph.D., Research Investigator, Endaural Hospital; New York City Fellow in Research, Harvard Medical School; formerly Assistant in Applied Anatomy and Pathology in Otolaryngology, Washington University Medical School, St. Louis, Mo. Pp. viii+1080, with 505 illustrations and 28 color plates. Baltimore, The Williams & Wilkins Company, 1947. (Price $18.00.)

Only once or twice in a decade a book appears which fills a great need and fills it so completely as to give promise of becoming a classic. This is such a book. More than one otolaryngologist has contemplated writing a histopathology of the ear, nose and throat and been confronted with two deterring facts: first, the histopathology of these special regions differs very little from general histopathology, and second, the newly widespread interest in the subject has resulted in such a mass of writing that a book might be in a fair way to being out of date before it saw the light.

The present authors have had the courage and the energy to turn out a work so complete and comprehensive and full of detail as to override such considerations.

Teacher, student and practitioner will find here all that could be desired. One consults it as a reference book and cannot lay it down. In 42 chapters it covers the entire scope of the ear, nose, pharynx and larynx.

There are more than 500 illustrations, many in color, and they are the authors' own, except in rare cases where they actually refer to the work of others. One may safely prophesy for this book the success which it richly deserves.

Diseases of the Nose and Throat.

By Charles J. Imperatori, M.D., F.A.C.S., Consulting Laryngologist, Harlem Hospital, N. Y., and Nyack General Hospital, Nyack, N. Y.; Consulting Bronchoscopist, Manhattan Eye, Ear, and Throat Hospital, N. Y.; Flower and Fifth Avenue Hospital, N. Y., and Riker's Island Hospital, N. Y.; and Herman J. Burman, M.D., F.A.C.S., Director of Department of Otolaryngology, Harlem Hospital, N. Y.; Instructor in Otolaryngology, College of Physicians and Surgeons, Columbia University, N. Y.; Assisting Visiting Otolaryngologist, Presbyterian Hospital and Vanderbilt Clinic. 3rd ed., pp. xxvi+576, with 480 illustrations, Philadelphia, J. B. Lippincott Company, 1947. (Price $12.00.)

This is a recent third edition of the well-known text which appeared for the first time in 1935. The book has been revised and brought up to date to keep pace with the newer conceptions in physiology, general nutritional states and specific therapy. Special attention has been given also to the section on radiation therapy.

The Subtle Sense.

By Ralph Bienfang. Pages ix+157. Norman, Okla., The University of Oklahoma Press, 1946. (Price $2.00.)

To those who enjoy an occasional digression from the strict routine of nasal physiology into the by-ways of the sensorium, this small and whimsical book will appeal.

While somewhat reminiscent of Dan McKenzie's "Aromatics and the Soul," it is by no means a counterpart. The nature of the book may best be characterized by a few of the chapter headings: "The Substance of Odor," "Odor and Food," "Odor in Society," "Odor as a Warning," "Odor and Literature."

Toward the end the author lets himself go and for four teeming pages sets down a list of such daily olfactory experiences of "depth-plumbing poignancy" as "the heavy oiliness of putty," "the light green narcoticity of ether," "the algal odor of a wet bathing suit," "the sickening off-cucurbitaceous character of a rotten melon," "the gross nitrogeny of a wet dog," "the conglomerate femininity of an opened purse."

Pellagra in the Oto-Neurology and Rhino-Laryngology.

By Otto L. E. DeRaadt. Pp. xvi+176, Leiden, Universitaire pers Leiden, 1947.

This monograph records the observations of the author while a prisoner of war in Java and Sumatra from March 1942 to October 1945. For three and a half years he served as ear, nose and throat specialist in a team of physicians and other medical personnel formed from the ranks of the prisoners. Their findings have been set down in great detail and constitute a valuable medical document.

Medicine in the Changing Order.

Report of The New York Academy of Medicine Committee on Medicine and the Changing Order. Pp. xviii+240, New York, The Commonwealth Fund, 1947. (Price $2.00.)

Five years ago The New York Academy of Medicine formed the Committee on Medicine and the Changing Order. The 48 committee members—laymen and experts from the fields of medicine, dentistry and nursing—have examined critically every phase of the current medical situation including medical education, nursing service, hospital facilities and medical research. This report draws together the findings of the Committee's study and its thoughtful conclusions as to what may best be done to remedy the present inadequacies.

Abstracts of Current Articles

EAR

Some Orientating Remarks on Tuning Forks and Audiometer and on the Registration of Defects of Hearing.

Mygind, S. H.: Acta Oto-Laryngologica 33:1, 1945.

An excellent and highly scientific article seeking to explain the apparent discrepancies between tuning fork tests and audiometric readings. The author states that the relation between the physical energy of the source of the sound and the biological loudness perceived is not the same for all frequencies. In conduction deafness there is a loss for low tones for tuning forks, while audiograms may show a horizontal line or even a drop at the higher frequencies. With the same addition in physical energy the increase in subjective loudness is greater in the lower range.

The accuracy of tuning forks and monochord is no less than that of the audiometer. Both methods are valuable and should be used intelligently.

HILL.

The Hereditary Character of the Cellular System in the Mastoid Process.

Dahlberg, G., and Diament, M.: Acta Oto-Laryngologica 33:378, 1945.

This is an interesting speculative investigation as to the possible part heredity plays in the type of pneumatization of the mastoid. The authors feel that the heredity factor is 1.56 times greater than the environmental factor, casting doubt upon Wittmaack's theory. This also is used to suggest the influence of heredity upon the development of chronic suppurative otitis.

HILL.

Loading and Covering of the Tympanic Membrane and Observation of the External Auditory Canal.

Van Deshoeck, H. A. E., and De Wit, G.: Acta Oto-Laryngologica 32:99, 1944.

Central covering of the ear drum gave no appreciable loss of hearing. Covering with fixation at the periphery caused deafness along the entire tonal range and loading resulted in deafness for low

tones. Various methods of covering were employed: cotton and vaseline, mercury, oil, water and collodion membrane. No difference was found between loading of the umbo, the pars tensa or the pars flaccida. Obstruction of the external auditory canal gives a loss of hearing for all tones. Partial obstruction causes loss for higher tones.

HILL.

The Functional Effect of Loading of the Tympanic Membrane.

Lüscher, E.: Acta Oto-Laryngologica 33:265, 1945.

The author feels that Van Deshoeck's and De Wit's repetition of his experiments confirm his findings: that experimental middle ear deafness, produced by various loadings of the tympanic membrane, produces varying audiograms. Some of these show a preponderant loss of low tones; others, of high tones. He feels that their disagreement with his conclusion that the pars tensa takes part in the transmission of tones of all frequencies, is based upon erroneous interpretation of the experiments.

HILL.

The Nature of the Acoustic Lesion in Acute Otitis Media.

Carlens, E.: Acta Oto-Laryngologica 32:453, 1944.

In acute otitis media due to pneumococcus, type III, there is marked disproportion in the hearing loss as compared to the slight changes seen in the membrana tympani. Deafness is predominantly of the perception type, due to toxic effect on the cochlea which is influenced by a poor antibody response in these patients.

HILL.

Sudden Complete or Partial Loss of Function of the Octavus System in Apparently Normal Persons.

De Kleyn, A.: Acta Oto-Laryngologica 32:407, 1944.

The author reports 21 cases of sudden loss of either acoustic or vestibular function, or both, which he attributes to some lesion of the central nervous system, probably vascular. In some of the cases there was a varying degree of screening; in others the damage was permanent. The author tends to think of Ménière's syndrome in

the original terms of hemorrhage and fails to give consideration to the more probable explanation found in increased intralabyrinthine pressure or hydrops.

HILL.

Acute and Chronic Scarlatinal Otitis.

Vaheri, E.: Acta Oto-Laryngologica 35:46, 1947.

In studying 2606 cases of scarlet fever, suppurative otitis media was found in 20.3 per cent. This was highest in the age group under five. Early onset of otitis was attributed directly to scarlatina while a later otitis was considered secondary to nasal infection. Mastoid operations were required in 9.3 per cent, the percentage being less in the younger group. Scarring of the membrana tympani, perforation or complete loss of the pars tensa were frequent results. Complete deafness was reported in 17 patients. Labyrinthitis is rare during the acute disease but may occur with chronic scarlatinal otitis.

HILL.

The Influence of Sulfonamides on Infectious Otitis Interna. Experiments with Guinea Pigs.

Sürala, V.: Acta Oto-Laryngologica 35:77, 1947.

In a control series, guinea pigs were infected with virulent hemolytic streptococci injected into the cochlea and into the middle ear. In those treated the infection was found to be checked, especially at points in the cochlea where there are natural obstructions and narrow spaces and where there is adequate blood supply. Treatment restricts the spread of infection and lessens the destruction of tissue.

HILL.

Fenestration Operation For Otosclerosis.

Passe, E. R. G.: The Lancet, pp. 171-174, Feb. 1, 1947.

Passe goes into the history of the operation noting that in 1876 Kessel removed the footplate of the stapes and reported a temporary improvement in hearing, that in 1897 Passow obtained a similar result by making a fenestra in the promontory, that in 1910 Bárány suggested making a fistula in the semicircular canal, that in 1914 Jenkins covered such a fistula with a Thiersch graft and that between the two world wars Holmgren and Sourdille did much of the

early work on this operation. He also reviews the contributions of Lempert. He goes into detail on the questions of the clinical diagnosis of otosclerosis, the clinical examination of the patient and the proper selection of cases for the operation. He states that not all cases of clinical otosclerosis can be improved by the fenestration procedure. For determining suitability for operation he divides his cases into three groups:

I. Those in which clinical and audiometric examinations for both pure tone and speech indicate that the residue of cochlear nerve function is adequate and in this group he expects 75 to 85 per cent success.

II. Borderline cases in which the damage to the cochlear nerve function is such that maximum improvement must be obtained to produce serviceable hearing. In this group success can only be expected in about 20 per cent.

III. Cases in which the cochlear nerve function has so far deteriorated that no chance of success can be anticipated.

Passe does not believe that the operation can be called successful unless the hearing can be restored to a practical level, that is, to 30 decibel loss or better within the range of 512 to 2048 d. v. Advanced age is not a contraindication to operation in his opinion if the cochlear nerve still retains its function. The ideal case, irrespective of age, is one in which the progress of the otosclerosis has been slow and of such degree that the cochlear nerve still retains an adequate amount of function. He states that if the hearing remains improved up to six months one can assume that the improvement will be permanent, although in a small percentage of cases the hearing may deteriorate up to two years.

Passe has performed 136 fenestration operations in all, in two series. Thirty-six were performed before the war with the fenestra in the lateral semicircular canal. In this series the improved hearing of all except two returned to its preoperative level or below. This poor result he lays to imperfect technique. Since August of 1945 he has performed another 100 operations and reports the results in 55 of these which were cases of six to twelve months' duration. In 34 of these 55 cases improvement was maintained for six to twelve months but he does not believe that final assessment of results can be made under two years. In all of these he has used the Lempert endaural technique.

GROVE.

NOSE

Acrylic Resin Implant For Atrophic Rhinitis (Preliminary Report).

Proud, G. O'Neil: Laryngoscope 57:256-262 (April) 1947.

In the past, the treatment of atrophic rhinitis has been no more successful than attempts to determine its cause. Conservative therapy has proven only palliative. Several surgical procedures have been devised to narrow the nasal airway. Lautenschlager and others accomplished this by infracting the lateral nasal walls. Success was contingent upon the formation of synechiae between the wall and the septum. Crusts diminished but nasal breathing was impaired and nasal physiology destroyed. Various types of implants have been inserted into the septum with some success. Failure is often due to extrusion or absorption of the implant. Proud reports the use of methyl methacrylate implants in seven cases with satisfactory results.

Proud's technique includes: septal resection with care in removing all cartilage chips, insertion of an acrylic implant which sufficiently narrows the nasal space but at the same time permits comfortable nasal respiration.

In six of the seven patients, crusting was diminished. In two of the three patients who had lost their sense of smell, this returned following the operation. In all patients with ozena, this distressing symptom was eliminated except in one case, where faulty technique caused failure.

Proud thinks that removal of the cartilage offers a solution to the old problem of prevention of extrusion of the implant. It is logical to assume that an implant is more apt to stay in place permanently when it lies between vital flaps of mucous membrane than when it rests in juxtaposition to cartilage which has been deprived of its supply of nutrition.

VAN ALYEA.

The Importance of Adequate Nasal Ventilation.

Treskow, Frank G.: Wisconsin M. J. 46:604-607 (June) 1947.

A perusal of this paper which is in reality a review of the physiology of the nose is well worth the time of any rhinologist. Treskow points out that the self-made diagnosis of "sinus trouble" with symptoms which include headaches, nasal stuffiness and dis-

charge, sore throats and symptoms referable to the ear are often dependent upon abnormalities in the normal physiological processes of the nose and more particularly, proper ventilation.

He calls attention to abnormal developments of the face so often associated with adenoids, with the resulting high-arched palate, septal abnormalities and a narrow nose. He stresses the importance of the proper position, direction and size of the naris in directing the stream of the inspired air and calls attention to its nozzle effect. He calls attention to a proper inspection of the nasal septum and notes the effect of its abnormalities in interfering with the direction of the air stream. These are deviations, spurs and abnormal thickenings. In the posterior portion of the nose the air current may be obstructed at its exit at the choana by thickenings of the free edge of the septum and hypertrophies of the posterior ends of the turbinates. He emphasizes the importance of a careful inspection of the nasopharynx in every individual with nasal symptoms, for the results of nasal pathology can frequently be observed on the posterior and lateral surfaces of the oropharynx and nasopharynx. He calls attention to the effects of air jets in changing the ciliated columnar epithelium into stratified squamous epithelium and describes the effects of this on the propulsion of the mucous blanket.

While a chronic progressive deafness cannot be ascribed to nasal blockage per se, poor ventilation can set up a chain of events which may lead to deafness due to the constant bathing of the eustachian tube orifices with irritating secretions.

There is nothing new in this presentation but the paper offers a good résumé of certain physiological facts concerning the nose, a knowledge of which will prevent the rhinologist from "going off the deep end."

GROVE.

Report of a Case of Recurring and Deforming Nasal Polyps.

Chatellier, H. P.: Les Annales D'Oto-Laryngologie 13:202-213 (May-June) 1946.

Report is made of a case of nasal polyps involving the frontal, ethmoid and maxillary sinuses on both sides. The case is of interest because of erosion of the bony partitions between the various sinuses and of the anterior wall of the frontal sinuses, resulting from the extreme pressure of the polyps. There was an external fistula in the frontal bone. The patient also presented broadening of the bony

framework of the nose from the pressure of the polyps. He had had nasal polyps for 20 years and had had them removed from the nasal cavity several times previously. The author performed external operations on all of the involved sinuses and states that the fistula closed. No follow-up on the patient is given.

HARKINS.

The Roentgen Treatment of Vasomotor Rhinitis.

Renander, A.: *Acta Oto-Laryngologica* 33:44, 1945.

A series of 221 patients with vasomotor rhinitis were given three treatments of 100 to 150 R at one to two day intervals. Freedom from symptoms was reported in 67 per cent, improvement in 23 per cent and no change in 10 per cent. It is considered that the effect is produced by nonspecific desensitization of the nasal mucosa.

HILL.

A Case of Mycosis in the Frontal Sinus.

Bergquist, B.: *Acta Oto-Laryngologica* 33:341, 1945.

Fungus infection of the frontal sinus is exceedingly rare. A case at first considered as acute sinusitis came to operation. This revealed a mass of mycelia, hyphomycetes. This was considered secondary to bacterial infection.

HILL.

Orbital Complications Following Operations on Paranasal Sinuses.

Godtfredsen, E.: *Acta Oto-Laryngologica* 33:211, 1945.

Orbital complications following ethmoid operations are more frequent and severe than those following operations on the antrum or frontal sinus. Usually these are orbital hematomata or lesions of the optic nerve. Orbitotomy may be necessary to save vision. When such a complication results, the cooperation of the ophthalmologist is essential. Unless operation is done within 12 to 20 hours after the lesion of the optic nerve is discernible, the prognosis is poor.

HILL.

Local Treatment with Sulfathiazole in Sinusitis.

Berdal, P.: Acta Oto-Laryngologica 33:245, 1945.

In a controlled series of cases the author did not find that sulfathiazole treatment reduced the number of required irrigations or the number of cases coming to operation. He considered its use of little or no therapeutic value and irritating to the mucous membrane.

HILL.

Nasal Diphtheria As the Cause of Septum Perforation.

Vaheri, E.: Acta Oto-Laryngologica 33:300, 1945.

Two cases of perforation of the septum apparently due to nasal diphtheria are reported. That perforation may follow atrophic rhinitis has been established. Likewise chronic nasal diphtheria may result in atrophic rhinitis. It is possible that nasal diphtheria may be the primary cause of certain cases of septal perforation.

HILL.

Hemolytic Streptococci in the Nose and Pharynx.

Skadhauge, K., and Kjau, M.: Acta Oto-Laryngologica, 33:145, 1945.

In a study of patients during a nonepidemic period it was found that 52 per cent of those showing hemolytic streptococci in the nose or pharynx following operation developed a postoperative fever as contrasted with 14 per cent of those not showing positive cultures. Complications in the first group were found in 15 per cent as against 3 per cent of the latter. The authors feel that whenever possible operation should be postponed until nose and throat cultures are negative.

HILL.

On the Genesis of the Dental Antrum Empyemas.

Moe, R.: Acta Oto-Laryngologica 35:42, 1947.

The usual route of dental infection of the antrum is through the root canal following a pulpitis. This may be difficult to diagnose from routine dental x-ray as the root apices may lie in the antrum and immediately below. X-ray films in several planes may be necessary as well as the routine vital tests for the suspected teeth.

HILL.

PHARYNX

Malignant Tumors of the Pharynx and Larynx.

Figi, Frederick A.: Wisconsin M. J. 46:611-614 (June) 1947.

Malignant tumors of the throat are encountered more frequently than benign lesions. Most malignant neoplasms of both pharynx and larynx grow rapidly. However they differ in their rate of metastasis. Those of the pharynx metastasize much earlier than those of the larynx. Because of the speed of extension malignant lesions of the pharynx are not amenable to surgery.

Oropharynx. Most malignant neoplasms of the pharynx are high-grade squamous cell epitheliomas or lymphosarcomas. Fibrosarcomas and adenocarcinomas occur here less frequently. The symptoms of cancer of the oropharynx may be severe, mild or entirely absent. As a rule epitheliomas produce a good deal of pain even in the early stages and this is often out of proportion to the clinical findings. This pain increases as ulceration takes place. Lymphosarcomata of this region are less painful and adenocarcinomata are often symptomless unless they attain considerable size.

Epitheliomas are not likely to be discovered until ulceration has taken place. However, infiltration of the lateral wall of the pharynx, the tonsillar region and the base of the tongue occurs without ulceration and in this type of case palpation with the finger is very important.

Lymphosarcoma usually produces a unilateral enlargement of the tonsil and moderate induration. Fibrosarcomas have a similar appearance but are more firm and localized and less liable to involve the tonsil.

Adenocarcinomas of the mixed tumor type are slow growing, painless, firm or hard and frequently nodular. They may be extensive and produce distortions of the pharynx and may be palpable externally. These tumors are classed as benign by some observers but as they recur if incompletely removed and as they may metastasize, Figi thinks this classification is unjustifiable.

If epitheliomas of the pharynx come under observation early, they may be electrocoagulated and this treatment may be supplemented by the implantation of radon seeds. In general, radiation is preferred to surgery in most of these cases.

Nasopharynx. Malignant tumors of the nasopharynx are chiefly malignant squamous cell epitheliomas and lymphosarcomas and orig-

inate most commonly in the fossa of Rosenmüller. They produce a characteristic symptom complex whose outstanding features are:

1. Deep-seated, aching or boring pain localized in the frontotemporal or parietal region, worse at night.

2. Symptoms referrable to the ear—tinnitus, pain and deafness.

3. Neurologic signs attributable to involvement of the second, third, fifth, sixth, ninth, tenth and twelfth cranial nerves.

4. The presence of a metastatic mass in the neck which may precede other symptoms by a number of months. The most common neurological symptom is a palsy of the external rectus muscle.

Because of the activity of these tumors and their surgical inaccessibility, irradiation of these tumors is the treatment of choice.

The rest of the paper is devoted to carcinoma of the larynx.

GROVE.

LARYNX

Reactions of Connective Tissue After Protracted Fractionated Irradiation of Laryngeal Carcinoma.

Windholz, Frank: *Radiology* 48:148-156 (Feb.) 1947.

The author describes and illustrates with 13 photomicrographs changes in connective tissue observed in the larynx one to ten months after protracted fractionated irradiation. This study was prompted by clinical observation of atrophy, sclerosis, edema, and necrosis of connective tissue in later stages of fractionated irradiation.

Atrophy of muscle fibers with increase in connective tissue, atrophy of fat cells and replacements by connective tissue, degenerative changes in elastic tissue are noted. Perichondrium and cartilage seem most susceptible and the changes may go on to abscess formation. Bone and periosteum, as a rule, show a greater resistance to irradiation. The author has found these histologic changes to be profound and extensive, and primarily and principally the same as those which appear after simple irradiation. The characteristic condition of the regenerated connective tissue is that of a hyaline sclerosis.

JORSTAD.

The Biological Significance of Fibrinous Radiomucositis of the Larynx.

Windholz, Frank: *Radiology* 48:157-160 (Feb.) 1947.

The significance of fibrinous radiomucositis of the oropharynx and larynx has never been definitely evaluated. The author studied a number of larynges (autopsy specimens) at or near the height of fibrinous mucositis. Microscopic examination conclusively shows that fibrinous exudation is not a reaction of tumor cells but of connective tissues and blood vessels. It is the mirror of connective tissue response to irradiation. Epithelial cells are unable to produce fibrin. Consequently, if we evaluate the clinical aspects of radiation therapy of carcinomas from the properties of radiomucositis in a particular case, we estimate the capacity of the connective tissue to participate favorably in radiobiological reactions, rather than the direct radiosensitivity of the cancer cells. It appears probable that connective tissue structures displaying strong fibrinous reactions are more likely to be transformed in the course of irradiation into a hyaline atrophic and fibrosclerotic condition than connective tissues which do not exhibit this property.

JORSTAD.

Late Changes in Mucous Membrane of the Irradiated Larynx.

Windholz, Frank: *Radiology* 48:274-281 (March) 1947.

The author has studied microscopically specimens of the mucous membranes of the irradiated human larynx one to ten months after protracted fractionated irradiation for carcinoma. Average tissue dose was 5800 r, Thoraeus filter, 200 kv., average rate 8.1 r/min., size of field 6 x 6 cm. By means of description and 12 photomicrographs there is demonstrated the radiobiological relationship of these late changes to the subepithelial connective tissue and to the retrogression of laryngeal carcinoma. The primary effect of irradiation is cell destruction. The mucous membrane is destroyed within 18 days. Regeneration of epithelium follows. This regenerated epithelium undergoes metaplasia. The resultant epithelial structures vary according to the condition of the subepithelial connective tissue. The author states these studies are the first conclusive proof and demonstration (histologic) of the interaction between sclerosis of connective tissue and atrophy of overlying epithelium.

JORSTAD.

Problems of Acquired Radioresistance of Cancer. Adaptation of Tumor Cells.

Windholz, Frank: *Radiology* 48:398-404 (April) 1947.

The author analyzes the causes of acquired radioresistance of malignant tumors on a cellular basis. The observations which enabled him to express an opinion were gained from histologic studies of the epithelium of the human larynx irradiated for cancer. The conclusions of Failla, drawn from in vitro experiments limited to experimental cancer strains, are similar to those of the author based on histologic studies on human tissue.

The various opinions as to the nature of this acquired resistance of malignant tumors following initial irradiation are summarized. Considerable evidence from these histologic studies is presented to show that the changes in radiosensitivity of the primary tumor, recurrent tumors or metastases are due to factors in the environment (cancer cell bed) rather than an inherent change in the cancer cells. The fate of the cancer cell in the new environment depends on its ability to adapt its metabolic and radiobiologic properties to those of the new host tissue.

JORSTAD.

Congenital Laryngeal Cysts in Early Infancy.

Ahlen, G., and Ranström, S.: *Acta Oto-Laryngologica* 32:483, 1944.

The authors review the symptomatology and treatment of these rare conditions, reporting one fatal case of their own. They suggest that the cysts are formed by a malformation in the fetal development of the laryngeal ventricle whereby the rudimentary cyst separates from the anlage of the ventriculus morgagnii.

HILL.

Surgical Treatment of Stenosis of the Larynx.

Aubry, M.: *Les Annales D'Oto-Laryngologie* 13:567-572 (Nov.-Dec.) 1946.

Presentation is given of the author's surgical technique in refractory cases of laryngeal stenosis. He first attacks the area below the glottis through a laryngostomy. Obstructing scar tissue is dissected out and a previously prepared tubular graft of skin is placed in the lumen, covering the raw surface, and the lumen filled with a mold of Stent's material. Three weeks later the supraglottic portion of the larynx is attacked. The cavity of the larynx is opened

through a vertical midline incision and scar tissue is dissected out. The hyoid bone is freed from its attachments throughout most of its length, through a transverse incision, and the freed hyoid is turned down and interposed between the two wings of the thyroid cartilage. The epiglottis is fixed to the hyoid by sutures. The lumen is filled with a mold of Stent's material. The laryngostomy wound and the transverse incision over the hyoid are closed. The molds are removed six weeks later and the patient is decannulated at that time.

The author reports the cases of three patients treated by this method, all having attained a good airway and a useful voice.

HARKINS.

BRONCHI

Bronchitis Circumscripta Nonspecifica.

Leegaard, T.: Acta Oto-Laryngologica, 33:391, 1945.

Hemoptysis, persistent cough and signs of bronchostenosis may be caused by a nonspecific inflammation resulting from acute catarrhal infection. This may cause secondary changes in the lungs, such as atelectasis, ulcerations, granulations and bronchiectasis. Tuberculosis, tumor and foreign body must always be excluded in the differential diagnosis. Six case histories are included.

HILL.

ESOPHAGUS

Cases of Familial Esophageal Stricture.

Aberg, S.: Acta Oto-Laryngologica 33:182, 1945.

This article describes a family in which nine members showed obstruction in the lower esophagus coming on in middle life. Four were examined by esophagoscopy and no tumors or specific inflammatory lesions were found. It would seem possible that the author was dealing with some form of cardiospasm rather than stricture.

HILL.

Congenital Cysts of the Oesophagus.

Ranström, S.: Acta Oto-Laryngologica, 33:487, 1945.

Two cases of congenital cysts of the esophagus are reported. Both were in the upper portion. The cyst wall was composed of partly ciliated cylindrical epithelium, tunica muscularis, submucous connective tissue and smooth muscle fibers in two layers. The author considers that these cysts develop from persisting vacuoles, surrounded by and detached from esophageal epithelium. These vacuoles are of normal occurrence during the second month of fetal life.

HILL.

On Idiopathic Cardiospasm and the Results of Treatment with Starck's Dilator.

Juul, A.: Acta Oto-Laryngologica, 32:85, 1944.

The author reports good or excellent results in 73 per cent of a series of 15 cases treated with the Starck dilator. He feels this method to be superior to the Plummer pneumatic dilator. The best results occur in cases where a distinct obstruction is felt which suddenly gives way on forcible dilatation, due to "rupture of the cardiac ring."

HILL.

MISCELLANEOUS

Medical Progress; Rubella (German Measles).

Wesselhoeft, C.: New England J. Med. 236:978 (Jan. 26) 1947.

In a review of the literature on rubella, Wesselhoeft devotes considerable attention to the congenital deformities resulting from prenatal infection with German measles. The lower virulence of rubella, as compared to other virus diseases, is less likely to result in miscarriage, the fetus is able to survive though there may be congenital deformities. Particularly because of the reports of Australian observers, considerable interest has been aroused in the occurrence of deafness in these cases. Apparently the severity of the rubella has no relation to the severity of damage in the ear. The defect seems to be in the organ of Corti. Many persons show islands of deafness. The fetus seems to be especially vulnerable during the first two months and evidence suggests that the chances are ten to one that gross congenital deformities will occur following maternal infection at this period.

HILL.

Effective Treatment of Extremely Sensitive Hay Fever Patients by the Chilling Method.

Lazarowitz, L. C.: J. Allergy 18:104-108 (Mar.) 1947.

The author calls attention to the fact that one of the chief disadvantages in the treatment of hay fever patients with hypodermic injections is the too rapid absorption of the antigen, especially if the patients are highly sensitive. This fact brings about some untoward reactions that may be serious at times and it also prevents the rapid desensitization of such patients because of the inability to arrive at a high enough dosage of the antigen to acquire a desensitization at an early date. He explains that in the past, various vehicles have been used, such as lanolin, olive oil, almond oil, glycerol, formalin, gelatin, and alum in an effort to slow up the absorption of the antigen, thereby preventing some of these reactions and making it possible to reach a high degree of desensitization earlier than would be the case if such vehicles were not used. In the majority of allergy clinics, the standardized aqueous ragweed extracts are used almost exclusively.

Lazarowitz has attempted to prevent this rapid absorption by chilling the area where the allergic extracts are to be injected. In a series of 30 cases, he has been applying an ice bag to the mid-outer part of the arm for 15 minutes before and 15 minutes following the subcutaneous injection of the aqueous ragweed extracts. The chilling method simply prevents the rapid absorption by creating a vasoconstriction and he has found that these patients can tolerate a much larger amount of ragweed solution when the injection is given this way than if given when the parts are not chilled. As a matter of fact, the local pseudopod-like wheal that would usually appear in from 4 to 22 minutes is delayed in its appearance to from 6 to 45 minutes when the ice bag is applied. He states that, roughly speaking, the ratio of 20:6 might be considered the quotient of the patient's tolerance to an increased dosage when handled by this plan.

Summarizing his work: of 30 patients one half were able to receive perennial treatment at long intervals so that the dosage could be rapidly increased to afford maximum protection during the season. The chilling before and after injections made it possible to do this without creating reactions even though the aqueous solution of the allergen was injected at intervals of as long as two or three weeks. This chilling procedure also is time-saving as it diminishes the number of visits by the patient to the allergist by 40 per cent. General constitutional reactions occurred very seldom and there were no de-

layed reactions observed. There was a greater degree of improvement in these individuals because of the ability to reach higher dosages at an earlier date. He also found that the chilling of the area made the injections painless.

McLAURIN.

Local Application of Penicillin in Oto-Rhino-Laryngology.

Piquet, J.: Les Annales D'Oto-Laryngologie 13:116-118 (March-April) 1946.

The author reports the successful use of penicillin solution (5000 units per cc.) locally in mastoid wounds after simple mastoidectomy, in three cases. The wounds were packed with gauze soaked in the penicillin solution, which was changed twice daily for five to eight days. There was prompt cessation of middle ear discharge and of drainage from the mastoid wound, and prompt healing of the wound. Two cases of acute maxillary sinusitis in children, one with osteomyelitis of the maxilla, were also treated with penicillin-soaked gauze packs in the sinuses, following operation. After eight days the patients were pronounced cured. Similar use of penicillin-impregnated gauze is reported in the treatment of one case of brain abscess, which was opened and drained in the course of mastoidectomy, with report of a cure.

HARKINS.

Mediastinal Emphysema and Pneumothorax as Complications of Tracheotomy.

Boysen, G., and Boysen, F.: Acta Oto-Laryngologica, 32:50, 1944.

Mediastinal emphysema may occur from direct injury of the pleura at operation, from disturbance of the thymus with indrawing of air into the mediastinum or from primary pulmonary emphysema with rupture of the alveoli from forced respiration. A case report is given.

HILL.

Paravertebral Anesthesia of the Plexus Cervicalis.

Hamberger, C. A., and Diament, H.: Acta Oto-Laryngologica 33:324, 1945.

In an investigation as to the cause of fatalities with paravertebral blood anesthesia, the authors carried out experiments in which they injected contrast material in cadavers and studied 78 cases of laryn-

geal cancer operated upon with this anesthesia. They conclude that if correctly executed, introducing the needle in a sagittal direction towards the transverse process and slightly posteriorly (Sorensen's method), it gives satisfactory anesthesia, but lethal complications may follow any deviation from correct technique.

HILL.

Spontaneous Postoperative Rupture of the Sigmoid Sinus.

Radmark, K.: Acta Oto-Laryngologica 33:63, 1945.

A boy of seven had spontaneous rupture of the sigmoid sinus eight days after operation. It was necessary to expose and ligate the transverse sinus to control bleeding. The author feels that it is unwise to uncover the sinus unnecessarily as it exposes it to undue risk, especially if it be wider than the opposite one, as shown on roentgenograms.

HILL.

On Encephalitis Otogenica Localisata Non-Suppurativa.

Richtner, N. A.: Acta Oto-Laryngologica 35:156, 1947.

This term is applied to a local, or more or less extensive inflammation in the brain secondary to otitis or mastoiditis. The symptomatology resembles that of brain abscess. Despite the author's plea for this to be recognized as a disease entity, it seems probable that it is the initial phase of what later may become a definite brain abscess, or, on the other hand, may resolve. His suggested therapy is sound in that he advises operation on the otitic focus with wide dural exposure, together with intensive chemotherapy. Further study, including possibly encephalography, is advocated before brain exploration is attempted.

HILL.

Penicillin In Intractable Asthma.

Miller, M. W.: J. Allergy 18:109-114 (Mar.) 1947.

Miller summarizes the present status of penicillin therapy in the treatment of asthma. He reports his experience in treating a total of 29 patients, 26 adults and three children. All of them had been thoroughly studied and treated from an allergic standpoint.

Bronchoscopies had been performed on 20 of the patients, including the three children, and autogenous vaccines made from the aspirated secretions were given.

After a year's effort to control the asthma by these methods with no improvement noted, penicillin was administered intramuscularly on the assumption that there must be some intrinsic etiologic factors which might be controlled. 12,500 to 25,000 units were given at three-hour intervals, with an average total dosage of 1,300,000 Oxford units.

Eleven of the 29 patients became symptom free or so greatly improved that further therapy was not necessary. Of these 11, seven continued to do well but four had recurrences to a lesser degree; these were given penicillin with further relief. Eleven more of the patients were moderately improved, but five of these continued to show symptoms which required other medication. The other six of this group relapsed promptly to their former status. Seven showed no change during the hospital course or thereafter.

The author noted that in these cases the symptoms were much more marked in the cold months and he found that the good results of treatment with penicillin were maintained longer in those treated in the spring than the fall. On this account he states that it is wise to follow each case for a period of at least a year before drawing any definite conclusions. He also states that in future it would be wise to study the penicillin sensitivity of the organisms found in the aspirated secretions.

McLAURIN.

Notices

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct examinations at the Palmer House, Chicago, Illinois, October 7 to 11, 1947. Communications should be addressed to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

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(List corrected to August 7, 1947)

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Otarion, Model A-1

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Otarion, Model A-3

Otarion, Model A-4 Jr.

Paravox Models VV2 and VV3

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Radioear Model 45-M-magnetic bone conduction receiver

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Sonotone Audicles No. 530 No. 531 and No. 533

Sonotone Model 600

Sonotone Model 700

Telex Model 22

Telex Model 612

Telex Model 900

Telex Model 1020

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Trimm Vacuum Tube No. 300

Unex Model "A"

Vacolite Model D

Western Electric Audiophone Ortho-Technic Model

Western Electric Telephone Type Audiophone, Model J-1

Western Electric Model 63

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Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11, Ill.

American Laryngological Association

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis 8, Mo.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: The Homestead, Virginia Hot Springs, April 14-15, 1948.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lyman G. Richards, 1101 Beacon St., Brookline, Mass.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Meeting: Chalfonte Haddon Hall, Atlantic City, N. J., April 7, 8, 9, 1948.

Sections:

Eastern—Chairman: Dr. J. Winston Fowlkes, 12 East 63rd St., New York 21, N. Y.
Meeting: New York, N. Y., January 16, 1948.

Southern—Chairman: Dr. William A. Wagner, American Bank Bldg., New Orleans 12, La.
Meeting: New Orleans, La., January 23, 1948.

Middle—Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Meeting: Columbus, Ohio, January 19, 1948.

Western—Chairman: Dr. Meade Mohun, 23 Second Ave., San Mateo, Calif.
Meeting: San Francisco, Calif., January 31-February 1, 1948.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Secretary: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich.

American Otological Society

President: Dr. Bernard J. McMahon, 634 N. Grand Ave., St. Louis 3, Mo.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.
Meeting: The Homestead, Virginia Hot Springs, April 12-13, 1948.